Paid Circulation, 30,920

The New England Journal of Medicine

FORMERLY THE BOSTON MEDICAL AND SURGICAL JOURNAL — *Established 1828*

VOLUME 251 • AUGUST 26, 1954 • NUMBER 9

Original Articles

Relation of Hospitals and Health Departments in Tomorrow's World 323
Charles F. Wilinsky

Endocardial Fibroelastosis: Factor in Heart Disease of Obscure Etiology 327
Wilbur A. Thomas, Raymond V. Randall, Edward F. Bland and Benjamin Castleman

Dislocation (Luxation) of Eyeball 338
Bertha Offenbach

Special Article

Letter from Indonesia 339
Raden Soeharto

Medical Progress

Proctology (Concluded) 343
E. Parker Hayden

Medical Intelligence

Abuse of Queckenstedt Test 348
D. J. LaFia

By the London Post 349
John Lister

Case Records of the Massachusetts General Hospital . . 350

Editorials

Public Welfare and the Physician 358
Administration Bills Rejected 359
Receding Tide 359

Massachusetts Medical Society 360
Massachusetts Department of Public Health Notices 362
Correspondence 362
Books Received 362
363

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2. Humphreys, P., *et al.*: Angiology 3:1 (Feb.) 1952.
3. Plotz, M.: New York State J. Med. *52*:2012 (Aug. 15) 1952.

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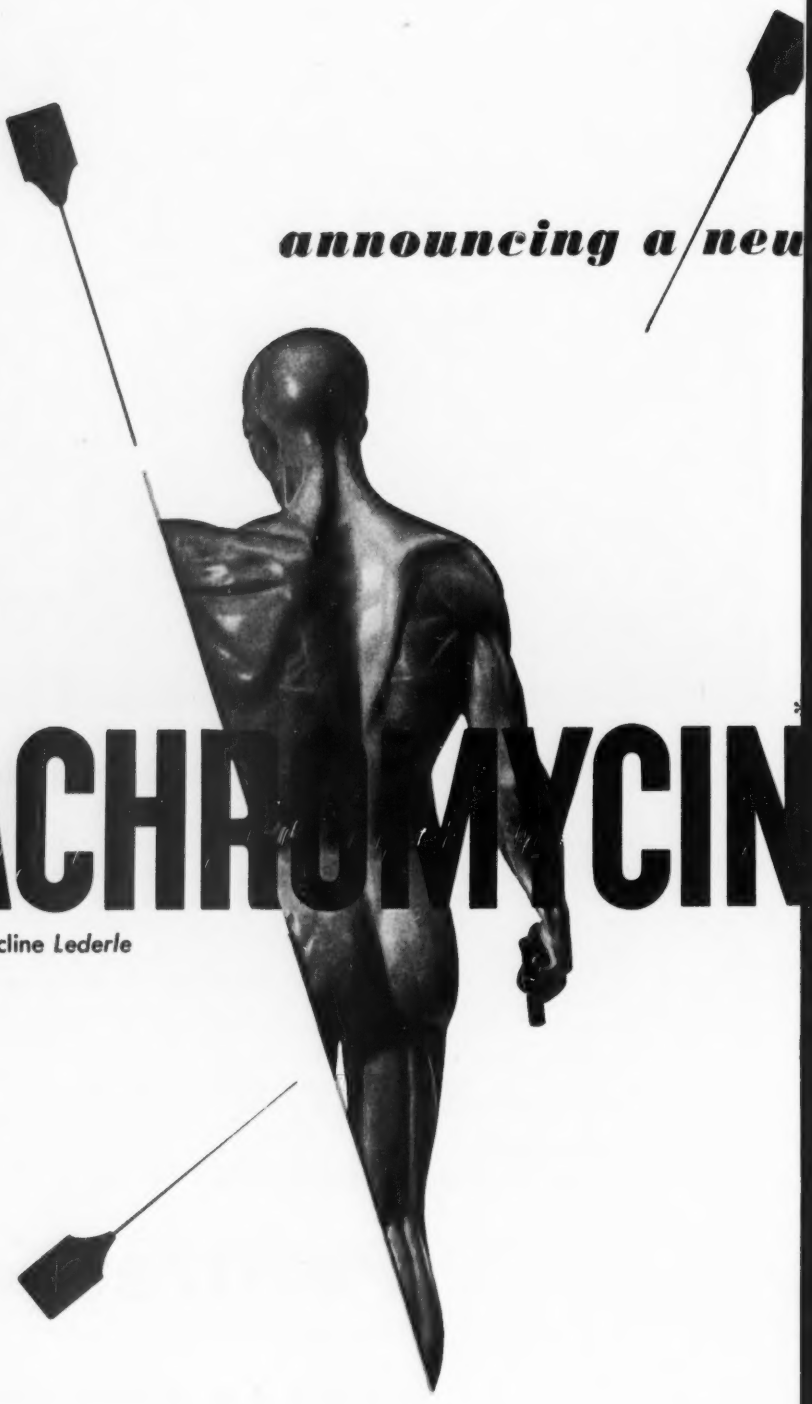

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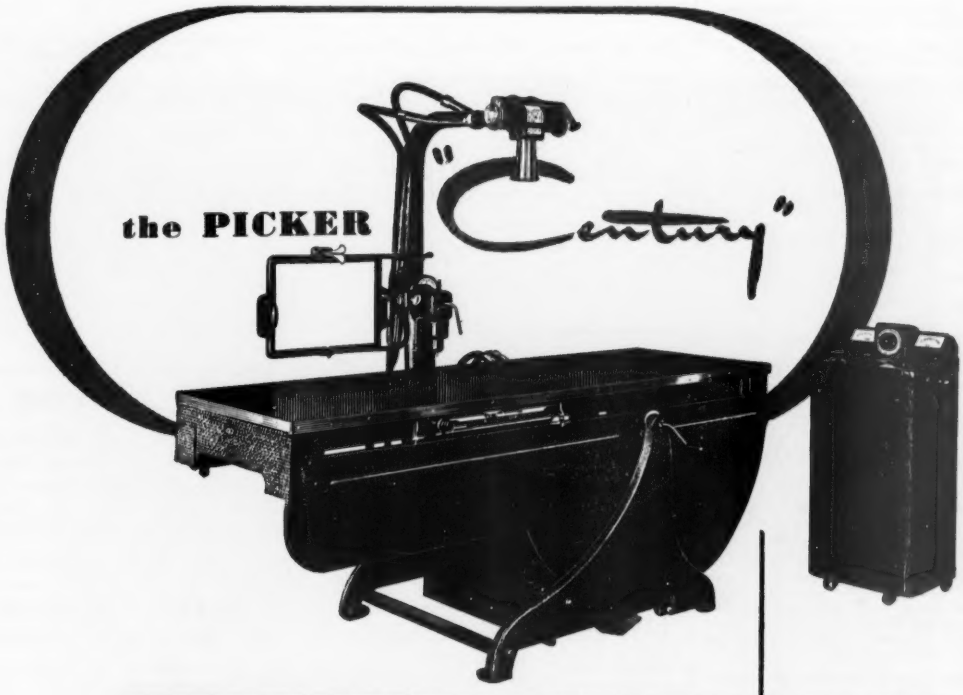

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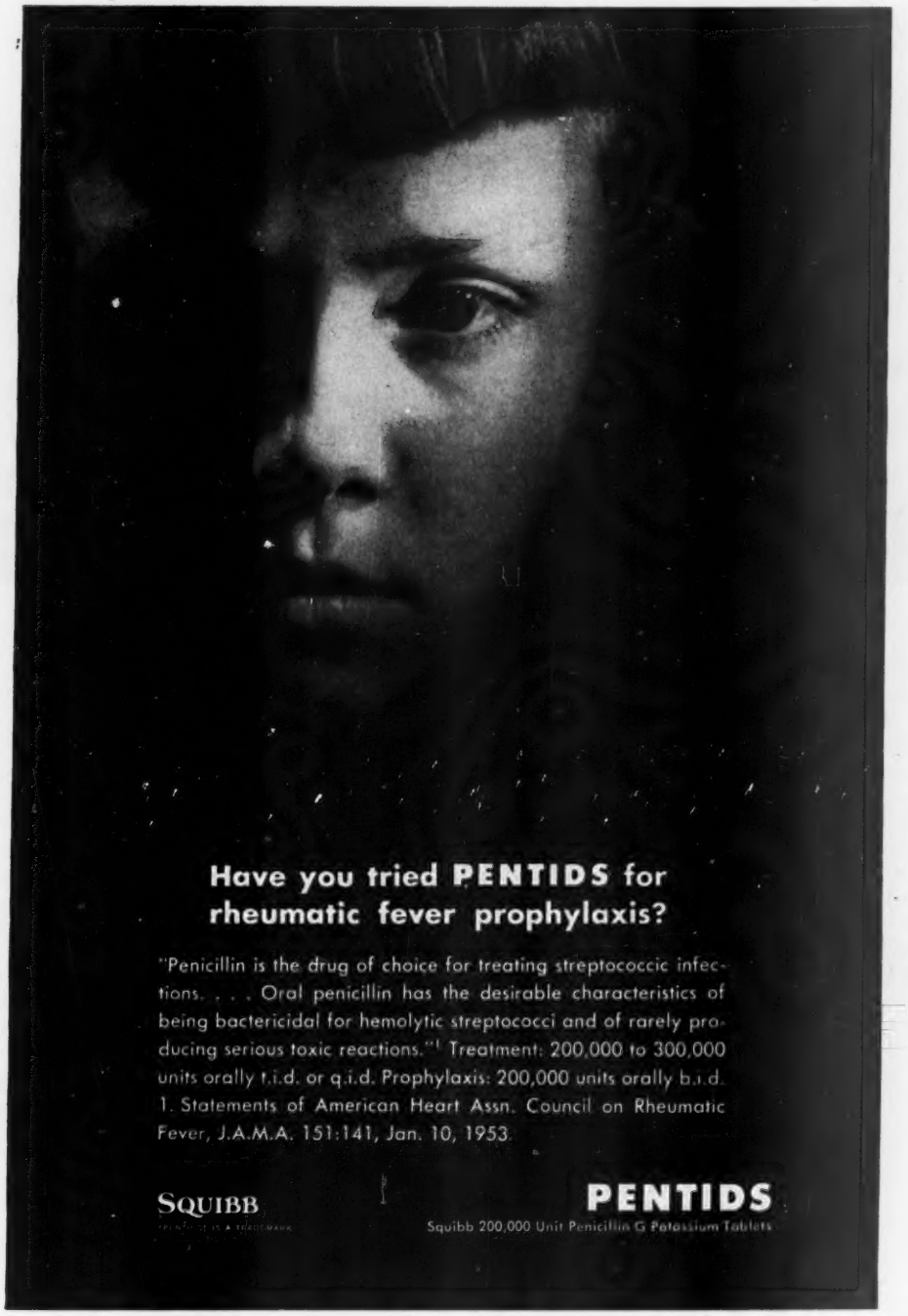
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Volume 251 AUGUST 26, 1954 Number 9

THE RELATION OF HOSPITALS AND HEALTH DEPARTMENTS IN TOMORROW'S WORLD*

CHARLES F. WILINSKY, M.D., Sc.D.†

BROOKLINE, MASSACHUSETTS

ALL who are concerned with the advancement of the public health derive comfort and satisfaction from the increasing interest in the furtherance of closer working relations between hospitals and health departments. Much has been said and a great deal has been written authoritatively on the values of this relation. Almost twenty years ago it was my privilege to serve as chairman of the Committee on Public Health Relations of the American Hospital Association, whose report to the hospitals of America made certain recommendations that hold true and are rich in values today. About five years ago the American Hospital Association and the American Public Health Association issued a joint statement of recommendations on the importance of co-ordination of hospitals and health departments.

When the worthwhile dreams of yesterday become the realities of today, one may be encouraged to believe that progress is taking place. Many of those who are interested in public health have been both intrigued and encouraged by the gradual narrowing of the gap between prevention and treatment of disease. They are happy, too, because of the increasing recognition of the sound belief that the cure of illness plays a significant role in the furtherance of the public health. It is appreciated today, as never before, that the challenging new health problems, which are an accompaniment particularly of middle and later life, —namely, cancer, heart disease, the arteriovascular lesions, diabetes, nephritis and so forth,—represent conditions in man that must become the concern of the vigilant health officer, and require the active participation and services of the physician and the hospital for their solution.

It seems fitting that the mutual interests and functions of health departments and hospitals be defined in the belief that an understanding of their responsibilities will prove the need for closer integration of effort on their part for the common good.

HISTORICAL BACKGROUND

One need not reach back into history thousands of years for gleamings of public-health interest but rather speak of the public-health program, as it is understood today, which dates back but a comparatively short time. Its reasonably modern foundation is historically related to the great sanitary awakening of a hundred years ago, ably described by Professor Winslow and representing the earnest pioneer efforts and stimulus of Chadwick and Simons in England. Theirs was the effort that brought about a better understanding of the relation between poverty and disease and of the complicating social factors involved in their solution. This sounds familiar indeed to students of what is termed "social medicine" today. In 1849, here in Massachusetts, from the roots of which stem many active public-health developments, Lemuel Shattuck wrote his historic report and made recommendations that have withstood the test of time; this report has been characterized by Professor Winslow as the most valuable document in the public-health literature. One is reminded that the part played by environmental sanitation in public health was soon followed by what has been so aptly termed the golden age of bacteriology stimulated by the epochal contributions of Pasteur and his contemporaries and successors.

The relation of these bacteriologic contributions to the work of the pioneer surgeon, Lister, his influence in furthering the principles of asepsis and the part this played in changing the character and concept of hospitals is familiar to students of public health and hospital evolution. This truly marked the dawn of a new day in the field of health and medical care, a renaissance responsible for the stimulation of probing minds to whom were opened wide gates of newer understanding of the causative factors responsible for the transmission of illness and what could be done for

*The Charles F. Wilinsky Lecture, presented at Harvard School of Public Health on May 14, 1954.

†Formerly, executive director, Beth Israel Hospital, and deputy health commissioner, City of Boston.

their control. The laboratory as a tool and invaluable aid came into its own. The story of water-borne, milk-borne and insect-borne diseases, as well as those directly spread by man, was learned. To this era in the history of mankind must be given credit for the rapid advances of the modern public-health movement.

Public Education

This was soon followed by a third development, described as the era of personal hygiene, which called into play the willingness of the people to avail themselves of discovered health measures and the participation and contributions of the sanitarian, the medical profession and the hospital. Methods for prevention, control and treatment of illness were available. Their acceptance and application were the great task. If the people were to have baby clinics or tuberculosis services or attempt to reduce the prevalence of disease by immunizing measures, they had not only to call on the skills and the disciplines of the medical profession but also to be willing to have these methods applied. Education of the public has played its part in advancing the theories and practices of Simon and Chadwick, Lemuel Shattuck, Pasteur, Koch and Lister and their collaborators. Comparative vital statistics offer the proof.

A hundred years ago the expectancy of life at birth was less than forty years. At the beginning of the present century this had increased to approximately fifty years. At present life expectancy is about sixty-eight years. At the beginning of the present century tuberculosis, a sensitive index of public-health progress, had a death rate greatly in excess of 100 per 100,000 population. A community today with a tuberculosis death rate in excess of 30 per 100,000 is not applying all the scientific knowledge at its disposal for prevention, control and treatment. Typhoid fever and diphtheria have been practically eliminated as causes of death wherever preventive methods have been applied.

At the beginning of the present century the infant mortality rate was in excess of 100 per 1000 live births. Today, it has been reduced in communities that apply available knowledge to approximately 25 per 1000 live births. For many years America occupied a very unenviable position in the maternal death rate. This has been much reduced in comparatively recent years. These are but some of the yardsticks of measurement of public-health progress in which education for the acceptance of these boons of mankind has become increasingly important.

Evolution of Hospitals

The development of hospitals in this country was not a rapid process until a comparatively short time ago. As recently as about seventy-five years ago there were 178 hospitals in the United States for the care of the sick with an approximate bed capacity of 35,000, of which a third were for the care of the mentally ill. Of this number only about 50 hospitals could be classified as belonging to the currently described voluntary, nonprofit institutions.

This contrasts with the present picture of about 6500 hospitals with more than 1,500,000 beds and serving 19,000,000 people annually, or about 1 out of every 8 of the American people. In this phenomenal growth asepsis, anesthesia and advances in surgery and in the medical sciences as a whole all played their part. More significant than this enormous expansion is a change in the character of the service rendered and the favorable reaction of the American people to the programs carried on in these hospitals. Today, hospitals represent what has been aptly characterized as "the workshops of the medical profession." They are citadels of health in which are grouped the skills of the medical profession, the availability of technical personnel, necessary scientific equipment and the laboratory, the x-ray department and the other services that make for diagnosis and treatment.

It is pertinent to allude to the investment in the hospitals of America, representing today the staggering sum of almost $8,000,000,000. It should also be pointed out that almost $4,000,000,000 a year is being spent for their maintenance. This capital investment for construction and equipment, as well as the cost of annual maintenance, is a tribute to the generosity of the American public, to those responsible for the expenditure of tax funds for the maintenance of these institutions and to the wisdom of the general public in recognizing these institutions as a most important factor in relation to their physical and mental well-being.

It is fitting to emphasize the total hospital functions. Although the primary purpose of the construction and equipment and organization of a hospital is the care of the sick, one must not lose sight of other exceedingly essential functions and their relation to individual and community well-being. I have in mind the importance of the hospital as an educational institution. The training of personnel, including the medical student and the house staff, and continuing medical education on a graduate level of its staff members, the training of nurses, of social workers and of technical personnel all represent educational functions that are important and related most intimately to the well-being of the public that these hospitals serve. The training facilities alluded to as a continuing educational process for the medical staff are reflected in the quality of medicine that the graduate practices. This educational program varies in type and size and in proportion to resources and needs. Every hospital, however, offers a background and material for the furtherance of needed education.

Research

Another exceedingly important part of the prevention, treatment and reduction of specific disease is the

maintenance of research. This activity may well be divided into clinical investigation carried on at the bedside of the sick and laboratory research in institutions with the financial resources that make this type of study possible. Another significant activity intimately related to the theme of this presentation is instruction in more healthful living. The degree to which this is carried on has in turn an increasing effect on the part of the hospital in the field of public health. Allusion to the wisdom of a program for better health calls attention to the routine contributions of the hospitals in their daily conduct of services of great public-health value.

Community Health

The prenatal clinic, the care of the expectant mother, adequate obstetric care and the care of the newborn represent public-health functions of utmost importance. The treatment of contagious disease and tuberculosis in institutions concerned, in a special sense, with the control of such threats to life and health is but one of the functions of hospitals. Allusion has already been made to management of illness as representing a public-health contribution, not only in the sense of direct cure of the specific condition but also in reducing the hazards of recurrence and complications. The value of rehabilitation is increasingly recognized in clinics and hospitals. Much has been learned about the worth of restoring to varying degrees the physical and mental usefulness of many whose potentialities, in the past, would have been ignored.

The growing concern of the health officer in the changing character of the practices of the medical profession and the hospitals in the years to come concerns illnesses that occur principally in middle and later life.

Allusion has been made to the increase in the expectancy of life and its influence on disease trends. Fifty years ago there were about 3,000,000 people over sixty-five years of age. Today, there are approximately 13,000,000. In the same period tuberculosis, formerly the leading cause of death, is in eighth position. Pneumonia has dropped from second to sixth place. Diarrhea and enteritis are no longer listed in the first ten causes of death, having been succeeded, in order, by heart disease, cancer, accidents, cerebral hemorrhage and nephritis.

This radical shift in the leading causes of death made possible by the programs of health departments and the skills of the medical profession compels careful readjustments of facilities to meet the future needs of an increasingly aging population. Many are involved in the planning for the future, including those responsible for medical education, hospital construction, organization and administration, as well as those concerned with the social and economic problems of elderly persons. Heart disease, cancer, nephritis, diabetes and cardiovascular changes represent the medical challenges of today and, in increasing measure, of the future. Of equal significance are plans to meet them best not only by adequate medical care but also by an integrated hospital program and an answer to social needs. This medical and social problem is of particular concern to the health officer of tomorrow. Although his interest in the programs of environmental sanitation and control of communicable disease and the furtherance of personal hygiene must continue, his activity in the field of geriatrics must increase. It is his responsibility that the necessary facilities for the solution of the newer health problems are available in the community. A vigilant, informed and participating medical profession, a balanced hospital program and the meeting of the required social needs to make these possible represent his interests and obligations.

The changing responsibilities of the health officer, on the one hand, and the hospital on the other call attention as never before to the necessity for integration of effort and the strengthening of the relation among the medical profession, hospitals and health departments. Recognition of this need by those who have given much serious thought to this subject is responsible for the development of a report, already alluded to, by the two national organizations (the American Public Health Association and the American Hospital Association) whose representatives saw the values of co-ordination of hospitals and health departments. This is why the Committee on Public Health Relations of the American Hospital Association, in 1936, stressed the following aspects of the problem:

> Newer conceptions of what constitutes an adequate community health program and the services which must be included in the same emphasize the gradual disappearance of the line of demarcation between the prevention and the treatment of disease. The changing point of view calls for greater unity and purpose and more intelligent co-planning and inter-relationship by the agencies engaged in the varied aspects of public health service. This has been expanded to include not only those programs which are basic for the control of mass public health but also facilities for the prevention and treatment of the many illnesses of man.

Those who labor faithfully and well in the hospital and public-health field appreciate the report of the American Hospital Association and the American Public Health Association calling attention to the following considerations:

> . . . among the foremost institutions which today serve the health of the community is the general hospital. Not only has the hospital developed during the past decades into the basic institution providing the technical facilities for adequate health appraisal and diagnosis and treatment of disease but it has also become an indispensable workshop for the practicing physician. Because of its important position the hospital's responsibilities and the total community health picture have been constantly expanding.

These documents, based on sound and serious thinking, emphasize the need for economy and closer inte-

gration, appreciate the needlessness of waste and duplication and make specific recommendations whenever they appear logically indicated. They recommend that hospitals and health departments be housed as a physical entity, particularly in rural areas, and call attention to the value of one laboratory and one x-ray unit to serve both hospital and health department whenever such an arrangement is justified. The baby, the prenatal, tuberculosis, venereal-disease, cancer and diabetic clinics, as well as those for crippled children, are but some of the activities in which the two groups have a common interest and may well be carried on jointly in the hospital or in its outpatient department. The use of the nurse or social workers of one agency for the conduct of the program of both may well be justified.

"Healthy minds in healthy bodies" is an old axiom. The vigilant health officer cannot divorce himself from his obligations to the field of mental hygiene; the good hospital cannot divorce itself from an interest in the mental health of the people that it serves.

Much has been said about the hospital as the health center of the community. It can only be characterized as deserving of this title when it carries on in addition to its accepted functions of the past a total interest in prevention, control and treatment of illness, and contributes as part of a team to the total health of the community.

The Future

What of tomorrow? The factors responsible for the remarkable reduction of preventable diseases are familiar, as is the effect of sanitation on water-borne and milk-borne diseases. The influence of laboratory practice, serums and vaccines on the prevention and treatment of certain diseases is appreciated. However, one cannot fail to re-emphasize the significance of the reduction of preventable disease on the increase of the diseases of middle and later life, which have already been alluded to and which the profession must be organized to care for.

These are troublesome times. The people of the world face critical decisions. Of one thing, however, one may be sure: the desire for good health is human and attainable. It is reassuring, therefore, to find that, in spite of the disastrous inferences that confront workers in public health, such a gathering as the present one has the laudable purpose of seeking better methods of organization and a more orderly process by which to meet the health needs of the people.

The horizons of hospitals and their staffs are broadening; expanding programs are being developed to meet the needs. I have in mind such additional services as home care, diagnostic clinics, clinics for the aged and group practice as weapons to be thoroughly studied in their relation to community requirements. In this study the medical profession, the health-department official, the hospital and its staff must work together for the good of all. Neither the hospital nor the health department as a community health service can afford to shut itself up in its own ivory tower and be content with the conduct of its own activities; it must play a part in community planning for the common good. This includes inter-relations with other hospitals and health councils, and close co-operation with governmental agencies, including departments of public welfare, as well as participation in the efficient planning of prepayment plans to the end that the burdens of unpredictable illness may be lifted from the shoulders of financially troubled men and women. There must be concern with the problems of the aged in relation to their medical care and other needs. Health education must be fostered so that the community will have a better understanding not only of the importance of health but also of the significant contribution of the physician, hospital and the health department to human welfare.

I should like to trace a pattern embracing what I believe to be the relation between the hospital and the health department in the world of tomorrow. Included in the pattern are the representatives of the medical and dental professions, the hospital, the health department, medical social workers and others concerned with human betterment and human welfare. Their combined planning should result in a plan to ensure the health protection required for organized society and functioning on a high level.

The challenging words of a great physician and distinguished public-health administrator, Hermann Biggs, have been borne out: "public health is purchasable and within certain limitations communities may determine their own death rates." This has been convincingly demonstrated by the lower morbidity and mortality rates in communities willing to expend funds for the purchase of health. An equally significant pronouncement—that "we are living in the century of the child"—is shown by the tremendous improvement in the field of infant and child health during the last fifty years. The hopes for better health call for the skills and the disciplines of the medical profession. The health problems of tomorrow, as I see them, must be solved in the offices of physicians and health departments and in outpatient and hospital facilities. Their solution will bring into play the additional skill of the social worker and others concerned with the total social picture. One cannot talk about better health without including such considerations as a job for the worker, adequate housing and sufficient and proper food. One must think in terms of an integrated hospital system that embraces the ambulatory patient, home care, the outpatient department, in-bed facilities of hospitals for acute illness, convalescence, rehabilitation and care of chronic diseases.

Conclusions

This discussion has been based on the realization that hospitals and public-health agencies have done

much over a period of years to contribute to better and happier living. Both groups have worked toward this objective, but greater efforts have been needed for the co-ordination of their activities. It is important to remember that in the past, hospitals as well as members of the medical profession have been regarded as concerned primarily with the care of the sick whereas health departments have been considered as agencies concerned only with prevention of disease. Allusion has already been made to the fortunately narrowing gap between prevention and treatment. In this, the medical schools have played a most significant part by the creation of departments of preventive medicine and developing a better understanding (in the mind of the medical student of today, who is to be the doctor of tomorrow) of interest and concern in both prevention and treatment. The need for this co-ordination of effort on the part of hospitals and health departments has become greater as the increase in the expectancy of life imposes a greater burden on curative machinery, placing the hospital in the strategic position of a vital agency in combating the illnesses of middle and later life.

Above all it must be remembered that this total program needs the support and co-operation of an intelligent public, trained to believe that public health is purchasable and is a desired goal for the men, women and children of America. The realization of this desirable goal imposes a challenging task on leaders in the fields of medical and public-health education, medical care, public-health practice and hospital administration.

ENDOCARDIAL FIBROELASTOSIS: A FACTOR IN HEART DISEASE OF OBSCURE ETIOLOGY*

A Study of 20 Autopsied Cases in Children and Adults

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BOSTON

EXTRAORDINARY advances have been made in the last fifty years in the diagnosis and classification of diseases of the heart. However, a small group difficult to fit into any well defined category remains, and each year scattered reports of isolated cases of obscure etiology appear.[1-11] These cases are often from special institutions restricted in age and sex distribution (such as children's and veterans' hospitals), and since the available material is at best diffuse and difficult to assemble, we have reviewed the total experience at the Massachusetts General Hospital during the past twenty-five years (10,000 autopsies) on heart disease of obscure etiology. The approach was by way of the autopsy files of the hospital, where all cases classified in the past as idiopathic cardiac hypertrophy, isolated (Fiedler's) myocarditis, endocardial fibroelastosis (without valvular involvement) or even atypical "beriberi" were restudied. Cases so classified but with evidence of any more clearly defined entities — namely, amyloidosis, hemochromatosis and glycogen-storage disease — were excluded. Furthermore, particular care was taken to exclude those with any clinical or morphologic indication of previous hypertension, valvular disease, significant congenital defects, cor pulmonale or coronary sclerosis.

The clinical and post-mortem records of these obscure cases were reviewed, the available gross specimens re-examined, and the microscopical sections studied. It quickly became evident that the most common and striking feature (in addition to hypertrophy) was the extensive fibroelastic thickening of the endocardium and of the subendocardial areas, and that this provided logical basis for a new classification and more detailed investigation. (Fig. 1, 2 and 3.)

In this series of 10,000 autopsies (since 1930) there were 24 cases of chronic heart disease with hypertrophy of uncertain etiology, and 20 of these had in common abnormal degrees of fibroelastosis. The remaining 4 cases had little in common with the above group or with each other. Two of these patients (forty-six and fifty-nine years of age) showed hypertrophy and dilatation without other distinguishing characteristics, and hence these cases must still be considered as truly "idiopathic." Another (a twenty-four-year-old woman) had no hypertrophy but a tremendously dilated, paper-thin right ventricle,[12] resembling in this respect the extraordinary "parchment heart" of an adult male patient encountered by Osler (about 1884) and subsequently recorded by Segall[13] (1950). The fourth patient (a twenty-four-year-old

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Supported in part by the Mickles Fund for cardiac research.

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woman) showed a severe giant-cell myocarditis. The remaining 20 cases of fibroelastosis (and hypertrophy) form the basis of this report. They have arbitrarily been divided on the basis of age and for purposes of

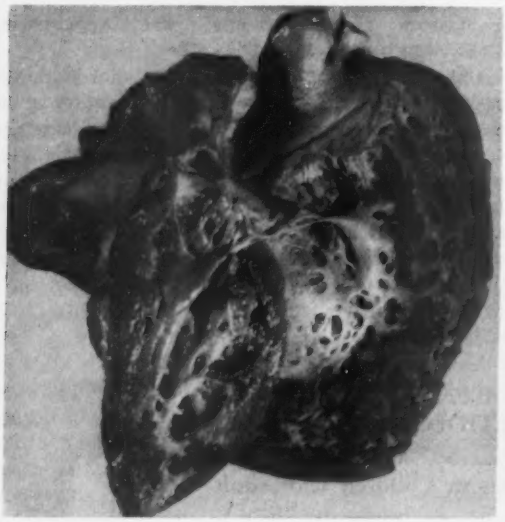

FIGURE 1. *Opened Left Ventricle in Case 6, Showing Gray, Opaque, Diffusely Thickened Endocardium with Mural Thrombi at the Apex in a Six-Year-Old Boy.*

discussion into three groups: infantile, childhood and adult (Table 1). The clinical course of each of these 20 cases is recorded below. Many of these cases, be-

FIGURE 2. *Left Ventricle in Case 17, Showing Diffuse Thickening of the Endocardium as in Figure 1, in a Fifty-Year-Old Woman.*

cause of their puzzling features, have been previously discussed in the Case Records of the Massachusetts General Hospital.

INFANTILE TYPE

All 4 patients in this group (under two years of age) gave evidence of severe cardiac dysfunction for at least several months before death. In 1 two-year-old infant the symptoms actually dated from birth.

The pathological features are presented in Table 1. All the patients showed marked cardiac hypertrophy, the weights varying from two to three times the expected normal. The only other striking feature was the presence of advanced generalized endocardial thickening, most prominent in the left ventricle. On microscopical examination this proved to be a prolifieration of both collagenous and elastic-tissue components (Fig. 4*A*). In most areas there was a clear line of demarcation between the thickened endocardium and the underlying myocardium. In some areas in 2 of the 4 cases there was patchy myocardial

FIGURE 3. *Left Ventricle in Case 19, Showing Widespread Patchy Thickening of the Endocardium in a Sixty-Three-Year-Old Man.*

fibrosis, which was occasionally located close to the endocardial surface. The myocardium was otherwise normal. There was no evidence of old or recent inflammation, and no fibrinoid necrosis in any of the sections examined. One heart showed mural thrombi in the left ventricle and infarcts in one kidney. A special effort was made to evaluate the status of the thebesian, arterioluminal and arteriosinusoidal vascular systems—an extremely difficult undertaking from a morphologic standpoint. We were unable to demonstrate any thrombosis of these vessels or other direct evidence of impairment. However, the fibroelastic thickening extended well into the sinusoids, and this no doubt interfered with the normal circulatory relations. It should be emphasized that the prominent feature in these cases was the generalized fibroelastic proliferation of the endocardium overlying normal myocardium in most areas. When myocardial fibrosis was present, it was of spotty distribution and, apparently, a secondary feature.

Endocardial fibroelastosis of this type in infants has received considerable attention in the medical literature in recent years, and evidence of a congenital ori-

gin has accumulated.[1-5] The presence of such severe endocardial proliferation and myocardial hypertrophy a case has been reported in a stillborn infant.[3] Hearts such as these, showing only mural endocardial changes,

TABLE 1. *Summary of Pathological Findings.*

CASE NO.	SEX	AGE	HEART WEIGHT gm.*	FIBROELASTIC THICKENING OF ENDOCARDIUM	MURAL THROMBI	ORGANS SHOWING EVIDENCE OF EMBOLIZATION	OTHER FINDINGS
Endocardial fibroelastosis, infantile type (under 2 yr. of age):							
1	F	3 mo.	68(23)	++ to +++			Globular heart, with +++ dilatation of lt. ventricle & lt. atrium; ++ patchy myocardial fibrosis; wall of rt. ventricle 2-4 mm. & that of lt. ventricle 2-8 mm. in width.
2	M	16 mo.	100(48)	+ to ++			No myocardial fibrosis; wall of rt. ventricle 4 mm. & that of lt. ventricle 9 mm. in width.
3[14]	M	2 yr.	125(56)	+ to ++			Generalized dilatation, especially of rt. atrium; no myocardial fibrosis.
4	M	2 yr.	100(56)	++	Lt. ventricle	Kidney & lung	Rt. & lt. ventricles moderately dilated; + patchy myocardial fibrosis; wall of rt. ventricle 3 mm. & that of lt. ventricle 9 mm. in width.
Endocardial fibroelastosis, childhood type (2-16 yr. of age):							
5[15]	M	5 yr.	150(85)	+ to +++	Lt. ventricle	Brain	Generalized dilatation, especially of rt. ventricle; + patchy myocardial fibrosis; wall of rt. ventricle 3 mm. & that of lt. ventricle 11 mm. in width.
6[16]	M	6 yr.	240(94)	++++	Rt. ventricle; lt. ventricle.	Lungs, spleen & kidney	+ patchy myocardial fibrosis; wall of rt. ventricle 3 mm. & that of lt. ventricle 11 mm. in width.
7[17]	M	7 yr.	210(100)	+ to +++	Lt. ventricle		Lt. ventricle dilated; ++ diffuse myocardial fibrosis.
8[18]	M	11 yr.	240(122)	++ to +++			All chambers dilated; no myocardial fibrosis; wall of rt. ventricle 3 mm. & that of lt. ventricle 15 mm. in width.
Endocardial fibroelastosis, adult type (over 16 yr. of age):							
9[19]	M	16 yr.	500	++ to +++		Lung	Enormous dilatation, especially on right; + patchy myocardial fibrosis; wall of rt. ventricle 6 mm. & that of lt. ventricle 15 mm. in width.
10[20]	M	21 yr.	600	0 to +++	Rt. atrium	Lung	All chambers tremendously dilated; no myocardial fibrosis; wall of rt. ventricle 5 mm. & that of lt. ventricle 14 mm. in width.
11[21]	M	21 yr.	750	++ to +++	Lt. ventricle	Kidney & lung	Moderate dilatation of lt. ventricle; + diffuse & ++ patchy myocardial fibrosis; wall of rt. ventricle 6 mm. & that of lt. ventricle 17 mm. in width.
12[22]	M	42 yr.	425	+ to ++	Lt. ventricle	Brain & lung	Rt. ventricle & rt. atrium twice normal size; lt. ventricle moderately dilated; no myocardial fibrosis; wall of rt. ventricle 5 mm. & that of lt. ventricle 15 mm. in width.
13	M	43 yr.	700	+ to +++	Rt. atrium		All chambers moderately dilated; no myocardial fibrosis; wall of rt. ventricle 7 mm. & that of lt. ventricle 15 mm. in width.
14[23]	M	44 yr.	340	+ to +++	Lt. atrium	Spleen & kidney	Rt. & lt. ventricles moderately dilated; + diffuse & ++ patch myocardial fibrosis; wall of rt. ventricle 4 mm. & that of lt. ventricle 10 mm. in width.
15[24]	M	46 yr.	450	+ to ++		Brain & kidney	Rt. atrium & rt. ventricle slightly dilated; + diffuse & ++ patchy myocardial fibrosis; wall of rt. ventricle 7 mm. & that of lt. ventricle 18 mm. in width.
16	F	49 yr.	380	++++	Rt. ventricle; lt. ventricle.	Legs, lungs, spleen & kidney	Globular heart, with slight dilatation of all chambers; no myocardial fibrosis; wall of rt. ventricle 4 mm. & that of lt. ventricle 15 mm. in width.
17[25]	F	50 yr.	490	+++	Rt. ventricle; lt. ventricle; rt. atrium	Lung, spleen & kidney	All chambers dilated; no myocardial fibrosis; slight coronary sclerosis.
18[26]	M	59 yr.	700	0 to ++	Lt. ventricle	Spleen & kidney	All cavities dilated; patchy area of dense myocardial fibrosis at apex of lt. ventricle; wall of rt. ventricle 9 mm. & that of lt. ventricle 17 mm. in width.
19	M	63 yr.	520	++ to ++++	Rt. atrium		Tremendous dilatation of all chambers; + patchy myocardial fibrosis; wall of rt. ventricle 8 mm. & that of lt. ventricle 13 mm. in width.
20	M	71 yr.	510	+++ to ++++			All cavities dilated; + patchy myocardial fibrosis; wall of rt. ventricle 4 mm. & that of lt. ventricle 12 mm. in width.

*Figures in parentheses represent normal weight for corresponding age.[28]

without evidence of inflammation in infants so young makes it almost certain to be of intra-uterine origin; have been linked with organs that, in addition, demonstrate overgrowth of the fibroelastic tissue involv-

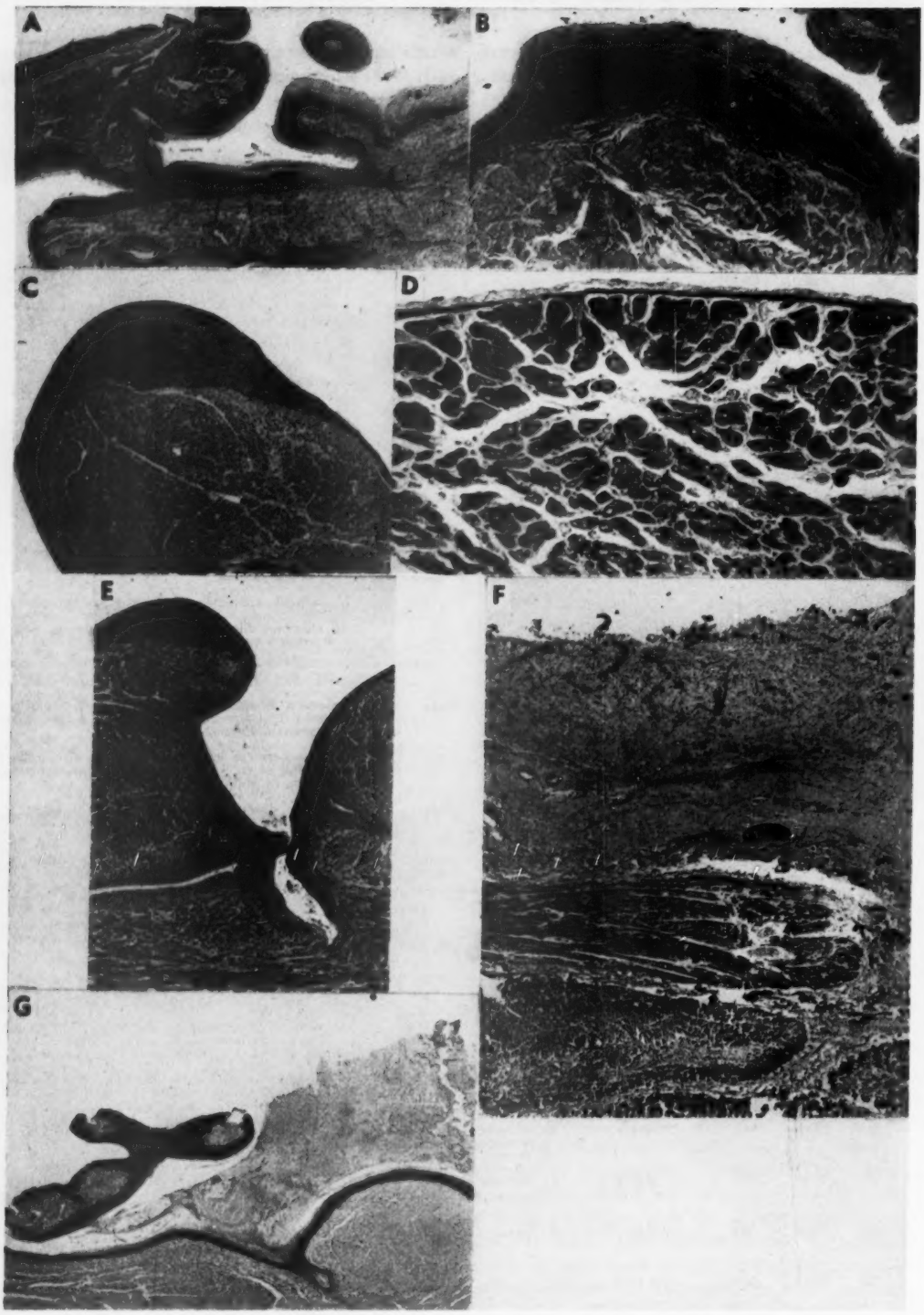

FIGURE 4. *Photomicrographs of Sections Prepared with Verhoeff's Elastic-Tissue Stain.*

A=(Case 1, aged three months), B=(Case 6, aged six years) and C=(Case 20, aged seventy-one years), showing fibroelastic thickening of endocardium; D=normal endocardium for comparison; E=(Case 13, aged forty-three years), showing fibroelastic thickening, with extension into sinusoids; F=inner half of wall of left ventricle in East African endomyocardial necrosis; the fibrous patch consists of organized mural thrombus and dense collagenous tissue replacing endocardium and adjacent myocardium (only a few broken elastic fibers remain); G=(Case 4) mural thrombus in which there is organization at base of thrombus, but elastic tissue of endocardium is intact in striking contrast to that shown in F.

ing one or more valves. Convincing evidence has been presented that this represents a further extension of the same process.[1] We have encountered a case of endocardial fibroelastosis in a two-day-old infant, with changes in the ventricular mural endocardium identical with those in the cases presented here and, in addition, mitral stenosis and aortic-valve deformity due to excessive proliferation of the endocardium of the aortic and mitral valves.*

It is beyond the scope of this paper to present all the facts in support of the congenital origin of infantile endocardial fibroelastosis. The entire problem of etiology has been thoroughly reviewed in a number of recent papers, and convincing evidence of its congenital origin brought forward.[1-5] Some authors have carried the matter further, and Johnson[6] has made the interesting suggestion that it results from intra-uterine endocardial anoxia due to premature closing of the foramen primum or foramen ovale at a crucial stage in development; we have found nothing that militates against this conclusion. The foramen ovale was closed in all our cases. The once popular theory of fetal endocarditis has been largely discarded because of the lack of histologic evidence of either healed or active inflammation.[11]

Childhood Type

The division of the childhood group (two to sixteen years of age) from the other two is arbitrary but convenient. Morphologically, these cases are identical with those of the infantile type except for a somewhat lesser degree of endocardial thickening and less striking hypertrophy (Table 1 and Fig. 1 and 4*B*). The only significant difference clinically was the later age of onset of symptoms. The clinical data are presented below.

This group is separated from the preceding one because of the reluctance of some observers to accept a common etiology. In spite of the morphologic and clinical similarities these investigators believe that the latent period between birth and the onset of symptoms militates strongly against the congenital theory of origin. However, this point of view is at variance with the well recognized freedom from impairment of patients with congenital heart disease who live normal lives for many years before the onset of cardiac symptoms.[27]

It is our belief that morphologic and clinical evidence strongly favors a common etiology in the infantile and in the childhood cases, and that the delayed appearance of impairment in the latter does not present a serious objection to this concept.

Adult Type

Cases of the adult type (over sixteen years of age) comprise a more controversial and puzzling group than the infantile and childhood cases, but similar terminology and classification are warranted on morphologic grounds. However, the etiologic aspects of this group require further consideration. All 12 patients had some degree of fibroelastic endocardial thickening, qualitatively identical with that in the preceding groups (Fig. 4*C, D* and *E*). In some it was as generalized and as severe as in any of the infant group (Fig. 2). In others it was less severe and tended to be patchy (Fig. 3). Myocardial fibrosis was no more prominent in these cases than it was in the infant group; in many it was entirely absent.

Mural thrombosis was a prominent feature in the adult group, and the resultant emboli often accentuated the clinical course. Nine patients had mural thrombi in one or more chambers. Eight of these, as well as 2 in whom no source was found, gave evidence of embolization in one or more organs.

Although painstaking search of the myocardium revealed a few lymphocytes in some cases these seemed insufficient to warrant serious consideration of an inflammatory origin and were no more numerous than those found in routine autopsy specimens.

Clinically, the average duration of symptoms was much longer than that in either of the preceding groups. Chronic congestive heart failure was more prominent, and a higher percentage of patients had evidence of embolization. Several patients gave a history of other members of the family who had died of heart disease but had not been studied post mortem. Detailed clinical descriptions of each case are given below, and a summary of the pathological data is presented in Table 1.

Etiologic Considerations

The morphologic similarity of these cases to those of infantile endocardial fibroelastosis is unquestionable. The congenital etiology of the infantile type is, we believe, equally unquestionable. It must be emphasized, however, that these conclusions do not necessarily commit us to the support of a congenital basis for the adult type. This assumption requires a careful examination and evaluation of the data that tend to support, but not to prove, the validity of the theory of congenital etiology. A point in its favor is the family history of obscure heart disease in close relatives. It is unfortunate that autopsies were not performed on any of the relatives in our series, but the point must be considered. Cases of infantile endocardial fibroelastosis in siblings have been reported.[5] No report has yet appeared of autopsies on adult siblings with the disease. The subject of "familial cardiomegaly" has recently been reviewed.[28]

Against a congenital etiology is the time lag between birth and the onset of symptoms, but here again we cite reports of cases in which autopsy reveals congenital heart disease in patients who have lived well into adult life before the onset of symptoms of cardiac impairment.[27] Moreover, most of the cases of endo-

*This case is not included here because of the additional complication of aortic and mitral stenosis.

cardial fibroelastosis in adults show a lesser degree of involvement than those in children, and it is therefore not unreasonable to suppose that they can live for a much longer period before symptoms become apparent.

Another aspect that must be considered is the lack of specificity of the endocardial fibroelastosis,[29] which is seen to some extent as a secondary feature in severe coronary sclerosis with either large or focal myocardial infarcts and is occasionally seen beneath the valve in aortic or mitral regurgitation. It is a constant feature in children who have an anomalous coronary artery arising from the pulmonary artery.[6] However, it is not a secondary feature of congestive heart failure, regardless of cause, as demonstrated by examination of several hundred hearts from patients dying with congestive heart failure. We did not see significant endocardial thickening except in cases with some clearly demonstrable cause for endomyocardial anoxia, such as considerable coronary sclerosis, or in those with severe valvular regurgitation. To answer completely objections raised on the grounds of lack of specificity requires the use of negative evidence, which is seldom as satisfactory as positive evidence. We have already stated that no cases were included in which there was clinical or morphologic evidence of any of the clearly defined forms of heart disease. Several less easily diagnosable diseases require special consideration.

Beriberi heart disease. There has been a tendency among many American authors to consider cases similar to those discussed above as examples of atypical beriberi.[7] This is a hypothesis that is difficult to support and equally difficult to disprove. Clinically, the diagnosis of beriberi heart disease is based on a history of dietary deficiencies coupled with a dramatic response to specific therapy. The only patients with this disease to be seen at autopsy are those who die before the treatment has had time to become effective and those who fail to respond to therapy. The autopsy findings in the former are entirely nonspecific, consisting largely of cardiac dilatation and myocardial edema.[30] The diagnosis in the latter is open to serious question, particularly if there is no history of dietary deficiencies. None of our patients gave a history of significant dietary inadequacies, and those who received vitamin concentrates showed no response.

Myocarditis. The possibility that these cases represent the end stages of some obscure infection has also been suggested. Saphir[31] and Gore and Saphir[32] have repeatedly demonstrated that myocarditis is an integral part of many types of infectious disease and can result in serious cardiac dysfunction. However, in none of these cases was there a history of an infectious disease that could reasonably be linked with the cardiac symptoms. One would have to assume that the initiating disease was so minor that it escaped attention, or that these were examples of isolated, or Fiedler's,[33] myocarditis. From the morphologic standpoint none of these hearts showed enough inflammatory infiltrate to warrant a diagnosis of myocarditis. In the cases with replacement myocardial fibrosis it might be argued that this represented the healed stage of a myocarditis, a possibility that cannot be entirely excluded. However, such fibrosis is also seen in cases of infantile fibroelastosis, and it seems more reasonable to assume that it is on the basis of anoxia. Furthermore, the myocardial changes are so overshadowed by the endocardial changes as to make it unlikely that they represent the primary lesion.

East African endomyocardial necrosis. In recent years a number of cases of a queer form of heart disease that is prevalent among East African natives have been reported in the literature. Clinically, the disease is similar to our cases of endocardial fibroelastosis.[34,35] There is evidence of marked cardiac dysfunction, manifested largely by unexplained congestive heart failure. From the pathological point of view, however, the diseases are distinctly different. We have been able to compare our specimens directly with hearts from 3 cases of endomyocardial necrosis that were sent to us by Professor J. N. P. Davies, of Makerere Medical College, East Africa. On gross examination the most prominent feature of his specimens is patchy endocardial thickening. However, on histologic examination the thickened areas show destruction of the original endocardium and adjacent myocardium, with replacement by vascular fibrous tissue. In these areas the elastic tissue of the original endocardium remains only as broken, irregular masses (Fig. 4*F*). This is in striking contrast to the intact proliferating elastic tissue in endocardial fibroelastosis (Fig. 4*G*).

"Collagen" disease. It has become popular to classify many obscure diseases under the ill defined term "collagen" disease. There was no evidence in the hearts or other tissues in any of these cases of a connection with any of the so-called "collagen" diseases, such as scleroderma, dermatomyositis, polyarteritis nodosa and lupus erythematosus. The changes in the endocardium were entirely proliferative, with none of the destructive lesions associated with the more common types of "collagen" disease.

There has been a recent report from South Africa[36] of 40 cases of unusual heart disease that the authors consider to represent a form of diffuse "collagen" disease. We have not had the opportunity to examine any of their material, but from the description and the photographs it appears that most of their cases are identical with those diagnosed as endomyocardial necrosis in East Africa. They have made an exhaustive histochemical and morphologic study and present data to support the thesis that endomyocardial necrosis is a new type of "collagen" disease. We cannot take issue with this portion of their report. However, elsewhere they make the following statement:

> In addition there is evidence to suggest that mild, but more generally distributed endocardial lesions may lead to

chronic diffuse endocardial sclerosis through a process of fibrillogenesis and elastic tissue hyperplasia. This may be an explanation of the rare examples of endocardial sclerosis in the adult.

They refer at this point to several reports in the literature, including the report of our Case 12, which appeared previously in the Case Records of the Massachusetts General Hospital.[22] They present no proof of this concept, nor can we present proof that it is incorrect. However, as stated above, our own observations indicate that endomyocardial necrosis and endocardial fibroelastosis represent distinctly different entities, and we have found nothing to suggest that our cases of endocardial fibroelastosis are a form of "collagen" disease.

Our opinion is that the clinical and morphologic similarity of the adult type of endocardial fibroelastosis to the infantile type strongly suggests a common congenital etiology. The frequent history of obscure heart disease in other members of a family lends support to this theory. There is no evidence as yet to disprove this theory of etiology, and no acceptable alternative has been presented.

Relation of Symptomatology to Pathological Findings

A problem that arises in cases of endocardial fibroelastosis in all age groups is correlation of the severity of the clinical symptoms with the relatively unimpressive morphologic changes. A number of theories have been advanced, and perhaps they all play a part. One theory is that the thickened endocardium interferes with proper contraction and relaxation of the heart in much the same way as a thickened pericardium does in constrictive pericarditis. Thus, this condition is sometimes called constrictive endocardial sclerosis. This theory seems to fit well with the facts, particularly in the cases in which there is marked generalized thickening. The clinical resemblance to constrictive pericarditis is sometimes so striking that on occasion an exploratory thoracotomy has been performed, as in Case 14.[23] However, none of these cases showed the contracted type of fibroelastosis with small left-sided chambers described in infants; on the contrary, most of the chambers were markedly dilated.

Another possibility is that the thickened endocardium interferes in some way with the proper conduction of contraction impulses. This theory is particularly attractive, since most cases show various arrhythmias or delayed conduction or both. However, it cannot be definitely substantiated until the histologists and physiologists determine the exact pathways for transmission of impulses through the heart.[37]

That the thickened endocardium interferes with the blood supply to the underlying muscle is another possibility. The presence of myocardial fibrosis in some of the cases lends support to this theory, but the absence of fibrosis in others, as well as its relatively minor degree in the remainder, suggests that this is not the only factor.

Case Reports

Case 1.* J.C., a 3-month-old female infant, was well until 4 weeks before admission, when anorexia developed. Two weeks later she began to have progressive dyspnea and cough. Physical examination revealed an acutely ill baby with dyspnea and faint cyanosis. The temperature was 98.6°F., the pulse 160, and the respirations 75 per minute. The blood pressure was 70/40. The heart was greatly enlarged, but no murmurs were heard. The entire left side of the chest was dull to percussion except for small areas at the apex and base of the lung. A roentgenogram taken the day before admission showed an enlarged heart. After admission the patient had increasing respiratory embarrassment. The temperature rose to 102.4°F., and she died several hours later. The clinical diagnosis was congenital heart disease.

Case 2.† P.Q., a 16-month-old male infant, had a generalized convulsion 6 weeks before admission. He was otherwise well until 2 weeks before entry, when grunting respirations developed. Three days before admission he began to have cough, anorexia and constipation. Physical examination disclosed respiratory distress. The temperature was 98.6°F., the pulse 140, and the respirations 50 per minute. The blood pressure was 95/65. The left eardrum and the throat were slightly red, and there were bilateral, shotty cervical lymph nodes. The heart was greatly enlarged. There was a gallop rhythm, but no murmurs were heard. The lungs were clear. The liver and spleen were not palpable. Examination of the blood demonstrated a red-cell count of 3,500,000, with a hemoglobin of 60 per cent (Sahli), and a white-cell count of 14,000, with 48 per cent neutrophils. A roentgenogram of the chest showed the heart to be markedly enlarged. On the morning after admission the respirations increased to 90 per minute, and the lungs filled with rales and rhonchi. The temperature rose to 101°F., and the patient died shortly thereafter. The clinical diagnosis was idiopathic hypertrophy of the heart.

Case 3.§ R.M., a 20-month-old male infant, was well until 5 months before admission, when he had whooping cough followed by pneumonia. Thereafter he had a cough, occasionally accompanied by vomiting. There was no fever. During the 3 weeks before admission he was restless and irritable. Physical examination revealed a chubby, pale infant who appeared chronically ill. The temperature was 101°F., the pulse 160, and the respirations 30 per minute. The throat was red, and there were shotty cervical lymph nodes. The heart was slightly enlarged, but no murmurs were heard. The lungs contained a few rales, and the liver edge was palpable at the level of the iliac crest. The white-cell count was 25,100, with a normal differential, and the red-cell count 5,800,000, with a hemoglobin of 72 per cent (Sahli). An electrocardiogram demonstrated an ectopic atrial tachycardia at a rate of 200 but was otherwise normal. A roentgenogram showed cardiac enlargement. After admission the patient had recurrent paroxysmal atrial tachycardia in spite of treatment with digitalis, acetylcholine, and injections of novocain into the sympathetic ganglions. Three and a half months after admission he had a brief episode of pneumonia. Five weeks later he had a second episode of pneumonia and died within 3 days. The clinical diagnoses were paroxysmal tachycardia and pneumonia of the left lower lobe.

Case 4.‡ P.S., a 27-month-old male infant, was admitted to the hospital because of cyanosis and dyspnea. Shortly after birth he had become cyanotic and was found to have an enlarged thymus. X-ray therapy was given, and the cyanosis disappeared. However, he seemed to be continually short of breath. Five weeks before admission progressive apathy, cough, dyspnea, vomiting, cyanosis and peripheral edema developed. Physical examination revealed dyspnea and cyanosis. The temperature was 96.0°F., the pulse 110, and

*Autopsy No. 8291.

†Autopsy No. 7459.

‡Autopsy No. 7247.[14]

§Autopsy No. 10,501.

the respirations 78 per minute. The blood pressure was 85/65. There were enlarged lymph nodes in the cervical region. The neck veins were distended, and there was slight pitting edema of all extremities. The left lung was clear, but a large hydrothorax was present on the right. The heart was enlarged. There were a gallop rhythm and a soft apical systolic murmur. The liver was moderately enlarged, and there seemed to be fluid in the abdomen. The white-cell count was 15,000, with 55 per cent neutrophils; the red-cell count, hemoglobin and urinalysis were normal. An electrocardiogram showed a sinus tachycardia at a rate of 140, right-axis deviation, notched P waves in Lead 1, prominent P waves in Leads 2 and 3, low voltage of the QRS complexes, depression of the ST segments and low or flat T waves. A roentgenogram demonstrated cardiac enlargement and hydrothorax on the right. The patient failed to respond to treatment, including 4 thoracenteses. He remained afebrile until the 16th hospital day, when the respirations became gasping and the temperature rose to 102.8°F. He died several hours later. The clinical diagnosis was heart disease of unknown cause.

CASE 5.* E. W., a 4-year-old boy, was admitted to the hospital because of a rapid heart. Two and a half years previously he had had his 1st episode of tachycardia. Subsequently, he had similar attacks about once a month that lasted from 20 minutes to 2 weeks. If an attack lasted more than 1 week edema of the face, hands and ankles usually developed. Physical examination revealed a pale but well developed boy. The temperature was 101°F., the pulse 90, and the respirations 24 per minute. The blood pressure was 92/52. The tonsils were moderately injected. The heart was slightly enlarged, and there was a soft systolic murmur over the precordium. The white-cell count ranged between 17,000 and 35,000, with a normal differential. An electrocardiogram showed a sinus arrhythmia with a rate between 50 and 70 per minute, a tendency to left-axis deviation and a QRS interval of 0.10 to 0.12 second. The P waves were prominent in Leads 1 and 2, and the T waves were low in Leads 1, 2 and 3. An electrocardiogram taken during a paroxysm of tachycardia disclosed ectopic atrial tachycardia at a rate of 280 per minute. While in the hospital he had several episodes of tachycardia. Acetylcholine and injections of novocain into the sympathetic ganglions were of no avail. He was discharged from the hospital after 5½ weeks.

Two weeks later there was an episode of tachycardia that lasted 14 days and then subsided spontaneously. Several hours later another attack started, and the patient returned to the hospital. The findings were unchanged from those of the previous admission except that the heart was normal in size, no murmurs were heard, and the liver was moderately enlarged. The tachycardia ceased suddenly on the 5th day after an injection of novocain into the right inferior cervical and upper dorsal sympathetic ganglions. Two days later the patient had a hemiplegia on the left and died within 18 hours. The clinical diagnoses were paroxysmal atrial tachycardia and cerebral embolism.

CASE 6.† L. O'C, a 6-year-old boy, was well until 3 weeks before admission, when constipation developed. Eight days before entry he had begun to have crampy, periumbilical pains and vomiting. Physical examination revealed a listless, thin boy with dyspnea. The temperature was 99.2°F., the pulse 120, and the respirations 40 to 50 per minute. The blood pressure was 90/60. The heart was moderately enlarged. There was a gallop rhythm, but no murmurs were heard. The liver seemed to be slightly enlarged. The white-cell count was 13,000, with 51 per cent neutrophils. The hemoglobin was 12.5 gm. per 100 cc. An electrocardiogram showed a sinus tachycardia, with a rate of 160, flat T waves in Lead 1 and inverted T waves in Leads 2 and 3. A roentgenogram disclosed cardiac enlargement, pulmonary congestion and fluid in both costophrenic angles. Three days after admission the patient complained of pain below the tip of the sternum, and the pulse rose to 160. He was given digitalis and oxygen. He improved, but on the 6th day a right hemiplegia developed. Ten days later he died after a 2d cerebrovascular accident. The clinical diagnosis was acute myocarditis of undetermined type.

CASE 7.§ K.G., a 7-year-old boy, began to have exertional dyspnea 5 weeks before admission, and 10 days later vomiting, diarrhea, pain in the right upper quadrant and rapid, grunting respirations developed. On admission to another hospital the heart was found to be enlarged. There was a faint apical systolic murmur, and the blood pressure was 80/60. The lungs were clear, but there was engorgement of the neck veins, hepatomegaly and facial edema. An electrocardiogram showed a sinus tachycardia at a rate of 110, a slightly prolonged PR interval and left-axis deviation. The patient failed to improve after 3 weeks of treatment with digitalis and mercurial diuretics and was transferred to the Massachusetts General Hospital.

Physical examination revealed a disoriented, dyspneic, cyanotic boy. The temperature was 95°F., the pulse 120, and the respirations 60 per minute. The blood pressure was unobtainable. The heart was greatly enlarged, but no murmurs were heard. The heart sounds were of poor quality, and there was a gallop rhythm. A pericardial friction rub was heard at the apex. There were a few moist rales at the lung bases, and the liver was moderately enlarged. The ankles were slightly edematous. The white-cell count was 24,800, with 88 per cent neutrophils. An electrocardiogram demonstrated a sinus tachycardia, at a rate of 150, and left-bundle-branch block. The patient became comatose and died 8 hours after admission. The clinical diagnosis was myocarditis.

CASE 8.‡ T.L., an 11-year-old boy, while playing outdoors, suddenly lost consciousness and died. At 5 years of age he had had a bowel upset, and on examination was noted to have slight cardiomegaly, tachycardia and a soft apical systolic murmur. He became dyspneic, and physical examination on admission disclosed distention of the neck veins, hepatomegaly and peripheral edema, but clear lungs. The heart was enlarged; there was a gallop rhythm, and a soft, apical systolic murmur was heard. An electrocardiogram showed a sinus tachycardia, left-axis deviation, a PR interval of 0.17 second, prominent P waves and low or flat T waves. A roentgenogram revealed cardiac enlargement. The patient was given digitalis, and the dyspnea, edema and hepatomegaly regressed. Although the apical systolic murmur disappeared, the heart did not regress in size. After 1 month the patient was greatly improved and was discharged.

During the subsequent 6 years before death he took digitalis. His activities were normal for his age, but the heart remained enlarged and at times a gallop rhythm was noted. On routine examination 1 month before death the pulse was regular, at a rate of 80 per minute, and the blood pressure was 95/65. The heart was moderately enlarged, but no murmurs were heard. An electrocardiogram demonstrated left-axis deviation, a PR interval of 0.20 second, prominent P waves and inverted T waves in Lead 1. The clinical diagnosis was idiopathic hypertrophy of the heart.

CASE 9.¶ T. O'K., a 15-year-old boy, was admitted to the hospital because of dyspnea. For the preceding 3 years he had been pale, apathetic and easily winded. Two weeks before admission he bought a bicycle and went to work as a messenger, and progressive dyspnea developed. Physical examination revealed a well developed boy with cyanosis. The temperature was 99.2°F., the pulse 150, and the respirations 30 to 40 per minute. The blood pressure was 110/90. The heart was moderately enlarged, and there were a gallop rhythm and a loud, blowing systolic murmur at the apex. There was tenderness in the right upper quadrant of the abdomen, but the liver edge was not felt. The lungs were clear, and there was no peripheral edema. Examination of the blood showed a red-cell count of 4,230,000, with a hemoglobin of 70 per cent (Sahli), and a white-cell count of 10,100, with 80 per cent neutrophils. An electrocardiogram disclosed a sinus tachycardia, low voltage of the QRS complexes, inverted T waves in Lead 1 and flat T waves in Leads

*Autopsy No. 7002.[28]

†Autopsy No. 13,035.[16]

§Autopsy No. 12,536.[17]

‡Autopsy No. 10,223.[18]

¶Autopsy No. 6158.[29]

2 and 3. A roentgenogram revealed enlargement of the heart. The patient was in the hospital for 48 days. During this time he was digitalized, and his symptoms regressed.

After discharge he was not at any time entirely well, and 11 months later he was readmitted to the hospital. The physical and laboratory findings were similar to those noted on the previous admission, except for the presence of a few rales at the base of the right lung and T-wave changes in the electrocardiogram to low in Lead 1 and slightly inverted in Leads 2 and 3. In spite of treatment with digitalis, salicylates and mercurial diuretics, the congestive heart failure increased, and he died 33 days after readmission. During the last 3 weeks the temperature intermittently increased to 100.6, and terminally it rose to 105°F. The clinical diagnosis was rheumatic heart disease.

CASE 10.* V.B., a 21-year-old man, was admitted to the hospital because of dyspnea. He had been well until 6 months before admission, when weakness, fatigue, dyspnea and progressive edema of the face, ankles, penis and scrotum developed. Two months before admission he had been studied in another hospital, where the heart was found to be enlarged but no murmurs were heard. The liver edge was palpable, and there was edema of the legs. During the month in the other hospital the temperature varied between 98.6 and 100°F., and he lost 28 pounds in weight. An older sister had died of a similar illness, and a younger brother was bothered by dyspnea and unexplained weight gain.

Physical examination revealed an obese, orthopneic man. The temperature was 100.2°F., the pulse 50, and the respirations 24 per minute. The blood pressure was 112/80. The heart was enlarged, and a soft systolic murmur was present at the apex. The lungs were clear, but the neck veins were distended. There was edema of the face, sacral area and ankles. The liver was moderately enlarged, and there was shifting dullness in both flanks. The venous pressure was equivalent to 250 mm. of water. An electrocardiogram demonstrated complete atrioventricular block and flat T waves. A roentgenogram of the chest showed cardiac enlargement and an area of increased density in the right costophrenic angle. After admission the patient became more dyspneic. On the 4th day he coughed up blood-streaked sputum and complained of pain in the left calf. The temperature rose to 102°F., and he died that evening. The clinical diagnosis was von Gierke's disease.

CASE 11.† R. C., a 21-year-old man, was admitted to the hospital because of tachycardia. One year previously he had had a sudden onset of diplopia, which lasted about 5 minutes. For 6 months he had had anorexia and occasional episodes of pain in the right upper quadrant of the abdomen. Three weeks before admission he had pain and swelling of the right elbow, which was relieved by hot pads. On the day of admission he suddenly fainted and was found to have an apical heart rate of 300 per minute. Physical examination showed a pale, well developed man with clammy extremities. The temperature was 99°F., the pulse 120, and the respirations 28 per minute. The blood pressure was 100/70. The heart was greatly enlarged. The cardiac sounds were obscured by noisy respirations, and no murmurs were heard. Numerous moist rales were present throughout both lungs, but the cervical veins were not distended, and there was no peripheral edema. The epigastrium was tender, but the liver was not palpable. The white-cell count was 19,800, with a normal differential, and the hemoglobin was 16 gm. per 100 cc. An electrocardiogram disclosed a ventricular tachycardia at a rate of 230, which, after the administration of Pronestyl, slowed to a sinus tachycardia at a rate of 120, with a PR interval of 0.22 second and right-bundle-branch block. A roentgenogram of the chest revealed cardiac enlargement and pulmonary congestion.

On the day after admission the patient began to expectorate bloody sputum, but additional roentgenograms of the chest showed no pulmonary abnormality except congestion. Treatment with cortisone was begun, and initially he seemed to improve. On the 7th day a gallop rhythm and a faint, apical diastolic murmur were noted, and the next day the liver appeared to be enlarged. On the 24th day he had a sternal ache and coughed up blood. A leukocytosis developed, and the temperature rose to 104.5°F. Both common femoral veins were ligated; no clots were found in the veins. The next morning he had another severe pain in the chest, and the blood pressure fell to 0. An electrocardiogram showed paroxysmal atrial tachycardia, with atrioventricular dissociation and a changing ventricular rate. A few hours later he again had a pain in the chest and died. The clinical diagnosis was idiopathic myocarditis.

CASE 12.§ H. O'C., a 42-year-old man, was well until 8 weeks before admission to the hospital, when intermittent pains high under the left lower ribs developed. The pains lasted only a few minutes and were associated with hurried eating or exertion. During the 8 weeks before admission he fainted on 2 or 3 occasions. Two weeks later orthopnea, vomiting and edema of the ankles developed. Physical examination revealed an acutely ill man with orthopnea, Cheyne–Stokes respirations and distended cervical veins. The temperature was 97.6°F., and the pulse 100 per minute. The blood pressure was 135/100. The heart was enlarged. There was a gallop rhythm, but no murmurs were heard. Rales were present throughout both lungs. The abdomen was distended with fluid; the liver was slightly enlarged, and the spleen was questionably palpable. The lower legs were edematous. An electrocardiogram showed slurring and low voltage of the QRS complexes in the standard leads, low T waves in Lead 1, slightly inverted T waves in Leads 2 and 3 and upright T waves in Lead CF_4. A roentgenogram of the chest demonstrated prominence of the left ventricle and a hazy mottling scattered throughout both lungs. On the day after entry the patient became restless and delirious. In spite of treatment he failed rapidly and died on the 5th day after admission. The clinical diagnosis was coronary-artery disease with thrombosis.

CASE 13.‡ J. M., a 43-year-old man, was well until 17 months before admission, when he had a sudden choking sensation. Subsequently, increasing dyspnea and orthopnea developed. Digitalis was given on several occasions but was discontinued because it seemed to cause dizziness and to aggravate the dyspnea. On one occasion he had a transient tachycardia (rate of 200 per minute) after the use of digitalis. Eleven years previously he had been rejected for military service because of suspected heart disease. No other details were available, except that the blood pressure was 145 systolic at that time. Physical examination revealed a chronically ill, dyspneic man. The temperature was 98.6°F., the pulse 106, and the respirations 22 per minute. The blood pressure was 120/95. The heart was markedly enlarged. There was a gallop rhythm, but no murmurs were heard. The liver was slightly enlarged, and the cervical veins were distended, but there was no peripheral edema and the lungs were clear. Electrocardiograms showed normal rhythm and the pattern of hypertrophy of the left ventricle. Digitalization was attempted and then discontinued because of dizziness in spite of which the patient's symptoms rapidly improved, and he returned home after 6 days.

Congestive heart failure again gradually developed, and he was readmitted to the hospital 6 months later. Physical examination disclosed a seriously ill man. The temperature was 97.8°F., the pulse 126, and the respirations 22 per minute. The blood pressure was 106/92. The heart sounds were distant, and there was a gallop rhythm. There were fine rales at the bases of both lungs and ascites and massive edema of the lower half of the body. An electrocardiogram showed a sinus tachycardia (rate of 110 per minute), hypertrophy of the left ventricle and left-bundle-branch block. Two hours after admission ectopic atrial tachycardia occurred, reverting to sinus rhythm after quinidine, and on the 6th day atrial fibrillation developed. He failed to respond to treatment and died on the 12th day. The clinical diagnosis was heart disease of undetermined etiology.

CASE 14.¶ J. S., a 43-year-old man, was well until 3 years before admission to the hospital, when dyspnea, edema and

*Autopsy No. 9844.[20]

†Autopsy No. 14,785.[21]

‡Autopsy No. 15,890.

§Autopsy No. 9009.[22]

¶Autopsy No. 11,365.[23]

weakness developed. He was treated for congestive heart failure and was well until the symptoms returned 1 year before admission. A younger brother had had similar symptoms, with cardiac enlargement, atrial fibrillation and an enlarged liver and spleen. Physical examination revealed a thin man who became dyspneic with exertion. The temperature was 98.6°F., the pulse 50, and the respirations 16 per minute. The blood pressure was 110/80. The skin was atrophic, and there was little subcutaneous tissue. The heart was slightly enlarged, and there was a moderately loud systolic murmur maximal at the lower end of the sternum and the apex. No rales were heard, but the bases of both lungs were dull to percussion. The cervical veins were distended, and there was moderate edema of the legs, sacrum and lower thoracic region. The edge of the liver was palpable at the level of the umbilicus, and the spleen was readily palpable. Examination of the blood showed a white-cell count of 10,200, with 78 per cent neutrophils, and a red-cell count of 4,610,000, with a hemoglobin of 13.6 gm. per 100 cc. The serum total protein was 4.4 gm. per 100 cc. An electrocardiogram showed an almost regular ventricular rate of 60 per minute, with frequent premature ventricular beats and no discernible atrial activity. There were low voltage of the QRS complexes, sagging ST segments in Leads 2 and 3, slightly elevated ST segments in Lead CF_4 and low or flat T waves in all leads. A roentgenogram of the chest disclosed moderate cardiac enlargement and hydrothorax on the left. The patient's condition remained unchanged in spite of treatment. On the 24th day an exploratory thoracotomy was negative for constrictive pericarditis. He was discharged 20 days after operation.

Six months later he was readmitted to the Massachusetts General Hospital. He had been somewhat improved until 6 weeks previously, when progressive congestive heart failure developed. One week before this admission he had had a sudden onset of headache, blurring of vision in the right eye and weakness of the left arm and leg. The findings noted on physical and laboratory examination were similar to those on the previous admission except for weakness of the left extremities, a bilateral hydrothorax and a grossly irregular cardiac rhythm, with occasional premature ventricular beats. He responded poorly to treatment. On the 20th day he complained of pain in the left side of the chest and 2 days later noted pain in the lower abdomen. He died on the 23d day after admission. The clinical diagnosis was cardiac hypertrophy, cause unknown.

CASE 15.* A. R., a 44-year-old man, was well until 3 years before admission, when he noted exertional dyspnea and fatigue. Nine months before admission he had undergone an abdominal operation and subsequently had had progressive congestive heart failure. Physical examination revealed a dyspneic man. The temperature was 98.3°F., and the pulse 65 per minute. The blood pressure was 100/65. The cervical veins were distended, and there was slight ankle edema. The heart was greatly enlarged, but no murmurs were heard. The anteroposterior diameter of the chest was increased, and the chest expansion decreased. Both lungs contained rales, and there were dullness and decreased breath sounds at both bases. The liver and spleen were not palpable. The white-cell count was 4200, with a normal differential, and the hemoglobin 12 gm. per 100 cc. An electrocardiogram demonstrated left-bundle-branch block. A roentgenogram of the chest showed cardiac enlargement, elevation of the right leaf of the diaphragm and a small hydrothorax on the right.

The patient responded slightly to treatment and was discharged on the 7th day. His condition remained much the same until 3 months later, when he suddenly experienced weakness of the left arm and leg and was readmitted to the Massachusetts General Hospital. On examination the findings were unchanged except for a partial left hemiplegia, from which he completely recovered in 10 days. He was discharged from the hospital 2 days later. He was fairly well thereafter until 5 months later, when he suddenly lost consciousness. He was taken to another hospital, where he regained consciousness in 48 hours. On the following day he was transferred to the Massachusetts General Hospital. Examination revealed a semicomatose man with a right hemiplegia. The temperature was 103°F., and the remainder of the examination was the same as on the previous two admissions. An electrocardiogram demonstrated an irregular rhythm at a rate of 70, a PR interval that varied from 0.10 to 0.34 second and left-bundle-branch block. Two days later an electrocardiogram disclosed atrial fibrillation. A roentgenogram of the chest showed the heart to be larger than previously, with pulmonary congestion and fluid in the right pleural space. The temperature returned to normal by the 5th day, but the paralysis persisted and his condition slowly deteriorated. He died on the 12th day after admission. The clinical diagnosis was myocarditis with a question of subendocardial fibrosis.

CASE 16.† E. C., a 49-year-old woman, was admitted to the hospital with embolic occlusion of the arteries of both legs and the left arm. A cardiac murmur had been noted 11 years previously, and in the 4 or 5 years before admission she had had progressive dyspnea and several bouts of mild congestive heart failure. Four days before admission she had a sudden pain in the left arm and a similar pain in the right calf the next day. Physical examination revealed arterial occlusion of both legs and the left arm. The temperature was 99.6°F., the pulse 100, and the respirations 22 per minute. The blood pressure was 100/90. The heart was slightly enlarged, and there were a soft apical systolic murmur and a short apical diastolic rumble. There were a few inspiratory rales throughout both lungs. Embolectomies were performed on the day of admission. On the 4th hospital day the patient had transient hemoptysis, and the next day abdominal distress and watery diarrhea. She did poorly and died suddenly on the 13th day after admission. The clinical diagnosis was rheumatic heart disease.

CASE 17.§ P. M., a 50-year-old woman, was well until 18 months before admission to the hospital, when progressive fatigue, exertional dyspnea, orthopnea and a weight loss of 65 to 70 pounds developed. Twelve months later she had had a febrile illness, allegedly pneumonia, which lasted 2 weeks, and in the last few days before admission she had several episodes of dyspnea and syncope. Physical examination revealed a poorly nourished woman with dyspnea, orthopnea and distended cervical veins. The temperature was 98°F., the pulse 100, and the respirations 24 per minute. The blood pressure was 114/80. The heart was markedly enlarged, and the cardiac sounds were faint, but no murmurs were heard. The lungs were clear except for a few rales at both bases, and the liver was moderately enlarged. The venous pressure was equivalent to 260 mm. of water. The circulation time, arm-to-tongue method, was 31 seconds. An electrocardiogram showed a sinus tachycardia, with an atrial rate of 105 and bigeminy from premature ventricular beats. There were slight right-axis deviation, low voltage and low or slightly inverted T waves. A roentgenogram of the chest demonstrated considerable cardiac enlargement and pulmonary congestion. The patient responded poorly to treatment. Shortly after admission tenderness in the left calf, a cough productive of blood-tinged sputum and a temperature of 100 to 102°F. developed. Both common femoral veins were ligated, and a course of Dicumarol was instituted. The low-grade fever and cough continued, and she grew weaker. She began to have episodes of severe dyspnea, and on the 39th day, 13 hours after the onset of such an episode, she died. An electrocardiogram 5 hours before death showed runs of ventricular flutter. The clinical diagnosis was cor pulmonale.

CASE 18.‡ J.K., a 59-year-old man, was well until 2 years before admission to the hospital, when he began to have exertional dyspnea. Six months before admission progressive orthopnea, edema of the ankles and abdominal swelling developed. Physical examination revealed a dyspneic, cyanotic man with distended cervical veins. The blood pressure was 120/80, and the pulse 80 per minute. The heart was moderately enlarged and there was a gallop rhythm, but no

*Autopsy No. 14,800.[24]

†Autopsy No. 15,370.

§Autopsy No. 15,601.[25]

‡Autopsy No. 13,982.[24]

murmurs were heard. There were dullness and decreased breath sounds over the base of the right lung. The liver was slightly enlarged, and there was ascites. Both lower legs were moderately edematous. The white-cell, differential and red-cell counts and the hemoglobin were normal. An electrocardiogram indicated normal sinus rhythm, with occasional premature ventricular beats and right-axis deviation. The QRS voltage was low, and there were nonspecific T-wave changes. A roentgenogram of the chest revealed cardiac enlargement, pulmonary congestion and a small amount of fluid in both costophrenic sinuses. The patient responded well to treatment with a low-sodium diet, digitalis and mercurial diuretics, and he was discharged on the 8th day.

Six months later he was readmitted to the Massachusetts General Hospital. He had been well until 3 months previously, when he began to have progressive edema. Three days before this admission a cough productive of small amounts of bloody sputum developed, and during the preceding evening he had a sharp pain in the right side of the chest. On examination the findings were similar to those noted on the 1st admission, with several exceptions. The temperature was 100.5°F., and the pulse 120. There were dullness, rales and decreased breath sounds over the base of the right lung. The white-cell count was 22,000, with 93 per cent neutrophils. An electrocardiogram was unchanged except for some increase in the voltage of the QRS complexes. A roentgenogram showed marked cardiac enlargement and an area of consolidation in the right lower lobe. In spite of treatment the patient failed rapidly and died 37 hours after admission. The clinical diagnosis was arteriosclerotic heart disease.

CASE 19.* S. G., a 61-year-old man, was well until 3 years before admission, when dyspnea developed and examination elsewhere showed cardiac enlargement. He was given digitalis, but the dyspnea became progressively worse. Physical examination revealed orthopnea. The temperature was 98.6°F., the pulse 88, and the respirations 28 per minute. The blood pressure was 145/85. There were a few moist rales at the bases of both lungs. The heart was slightly enlarged, and there was a slight systolic murmur at the apex. An electrocardiogram disclosed occasional runs of bigeminy and right-bundle-branch block. The patient was discharged after 2 days of observation.

In the subsequent 2 years the congestive heart failure increased, and he was seen repeatedly in the Out-Patient Department, being admitted to the hospital on 4 occasions. He responded poorly to treatment and died of congestive heart failure. The clinical diagnosis was arteriosclerotic heart disease.

CASE 20.† J. S., a 65-year-old man, was admitted to the hospital because of hematuria of 3 days' duration. He had always been well otherwise except for a "heart attack" 25 years previously, which had kept him in bed for 2 weeks; no further details of this illness were available. Physical examination showed a temperature of 97°F., a pulse of 84 and respirations of 20 per minute. The blood pressure was 130/90. The heart was moderately enlarged, and there were many extrasystoles. Cystoscopic examination demonstrated a low-grade malignant bladder tumor, which was removed. The patient made an uneventful recovery. He remained in good health for 2 years but then began to have dyspnea associated with chest pain. These symptoms continued in spite of treatment with digitalis.

Three years after the 1st admission he was readmitted to the hospital because of an acute febrile illness, thought to be infectious mononucleosis. His cardiac status remained unchanged. Eight months later he had an episode of severe chest pain, which confined him to bed for several weeks. Progressive congestive heart failure subsequently developed, and he was again admitted to the hospital, 2 years after the 2d admission. He died on the 3d hospital day. The clinical diagnosis was coronary-artery disease.

SUMMARY AND CONCLUSIONS

Twenty cases of heart disease of obscure etiology with the pathological finding of endocardial fibroelastosis are presented. The cases are divided arbitrarily into three age groups: *infantile* (under two years of age), *childhood* (two to sixteen years of age) and *adult* (over sixteen years of age). Evidence is presented that the cases in the infantile group are of congenital origin, that those in the childhood group are identical with those in the infantile group except for the later age of onset of symptoms, and that it is likely that they also are of congenital origin. A congenital etiology is proposed for the adult group, in spite of the lack of specificity of fibroelastosis and the time lag between birth and the onset of symptoms, because the hearts are morphologically identical with those in the earlier age groups.

Other unusual types of heart disease, including beriberi, myocarditis, East African endomyocardial necrosis and South African cardiovascular collagenosis, are discussed, and evidence that they represent different entities presented. The relation of the symptomatology to the pathological changes in endocardial fibroelastosis is discussed. Theories considered are interference with normal contraction by the thickened endocardium corresponding to that produced in constrictive pericarditis, impairment of conduction and myocardial anoxia caused by interference with vascular connections of the underlying endocardium.

*Autopsy No. 16,231.

†Autopsy No. 15,862.

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DISLOCATION (LUXATION) OF THE EYEBALL

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BOSTON

THIS condition is part of a general physical picture that can be managed most adequately by the general medical practitioner. Also, it can occur during a general examination, and its prevention should be a concern of every practitioner.

Luxation of the eyeball is defined as a forward displacement of the eye so that the lids close behind, not in front of, the eyeball.

CASE REPORT

A 38-year-old man who owned and operated a food store had been seen 2 years previously, when he weighed 250 pounds but had no notable disease. His recent visit showed central corneal ulcers. As the ophthalmologist separated the lids to instill drops the right eye slipped forward in front of the lids, dislocating completely (Fig. 1). The patient had no perceptible pain reaction. The eye was quickly replaced with slight but determined pressure on the sclera. The vision had not changed.

Forty-eight hours later, when the ulcers had healed, the right eye was deliberately proptosed. The patient stated that his eye felt as though it were falling out but was not painful. On repeated visits the right eye was dislocated at least 6 times. The left eye was displaced, but less easily. There was no change in his vision, which was corrected to 20/20. The refractive error was 4 diopters of hypermetropia. An internist reported the basal metabolic rate as −3 per cent. The ophthalmometer measured 25 mm. for the right eye and 23 mm. for the left. X-ray studies of the orbit showed normal measurements for depth, height and width.

The patient was put on a weight-reducing diet and lost 25 pounds in 3 months. The exophthalmos now measures 22 and 20 mm. Luxation of the right eye is still possible with considerable force. The left eye is firmly settled in its orbit. The lids are unusually flabby. The dislocation is considered to be due to excess adipose tissue in the orbit.

DISCUSSION

How does this luxation or complete proptosis come about? The factors that operate to hold the eye in the orbit—the lids, Tenon's capsule, extrinsic muscles and adequate space to hold the eyeball and the accessory organs—are overcome by lack of orbital space or shallow orbits, tumors or fluid in the orbital cavity, panniculus adiposus or pressure on the jugular veins when the head is held below the body with the eye open or holding of the breath so that the intraorbital pressure is increased. There are also anomalies of the muscles that are protractors opposing the backward pull of the recti muscles.

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The patient described above undoubtedly had an adipose mass displacing the eyeball forward as well as pressing against the orbicularis muscle, contributing to the weakness of the lids and helping in the dislocation, which was also promoted by the general pathology of

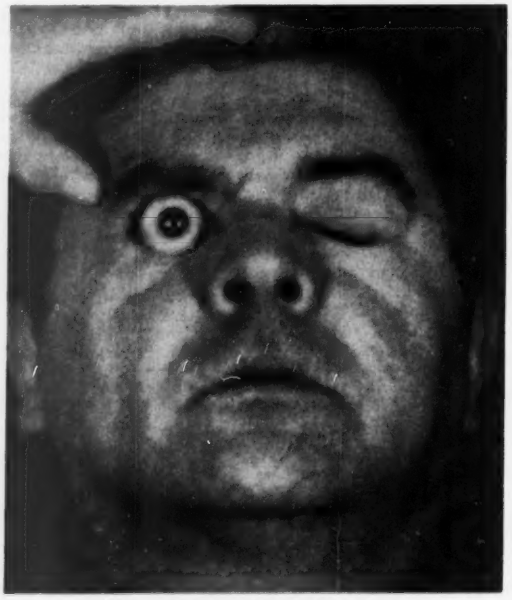

FIGURE 1. *Typical Appearance of a Patient with Dislocation of the Eyeball.*

adiposity. The fat infiltrated smooth muscle, causing degeneration of that tissue—in much the same way as fatty degeneration of the striated muscle of the heart occurs. According to Whitnall[1] involuntary musculature in the orbit forms an incomplete ring about the globe, connecting closely with Tenon's capsule. Also, palpebral involuntary muscle forms a thin sheet in the lid, connecting with the peripheral margin of the tarsal plate. These are additional factors in the displacement of the eye.

It is suggested that the case reported by Ruedemann, Roberts and Seligson[2] in a 300-pound man was also one of luxation due to adiposis within the orbit.

There are several types of dislocation according to

the etiology. The spontaneous cases are associated with shallow orbits, laxity of supporting tissues,[3] tumors (most often hemangioma[4]) and panniculus adiposus as in the case reported above. Spontaneous luxation has occurred in infants with oxycephaly and craniostenosis. Displacement takes place during crying or straining. The mechanism is probably increased congestion of the orbital tissues. Other precipitating factors are coughing, sneezing, bending down and separation of the lids. Ralph Lloyd[5] states that pressure on the opposite jugular vein will cause the eyeball to jump forward.

Incidentally, the veterinarian reports that the Pekingese dog has prominent eyeballs owing to the largeness of its eye. It is a belligerent animal and is often involved in fights that terminate in a luxated eyeball.

Voluntary dislocation in human beings has been described in the literature. This occurs through the contraction of oblique muscles or with muscle anomalies. In such a case studied at autopsy[6] an additional oblique muscle, which acted as a protractor, was found. Furthermore, the optic nerves were somewhat longer than normal, and the muscle insertions were farther back on the eyeball. It is reported that the fakirs of Rafae do penance after long practice by voluntarily propelling their eyeballs from the orbits.[7] "Believe-It-Or-Not" Ripley collected a case of a voluntary luxator who made the rounds of the circuses and fairs.

Luxations from trauma have been reported in the newborn, the delivery forceps thrusting the eyeball from the orbit. Avulsions have occurred during wartimes—as accidents during blackouts and on the battle front.

Finally, how does one manage the dislocated eyeball? Pressure on the sclera has been most effective. To relax the orbicularis warm compresses are considered successful. If the spasm becomes severe, canthotomy or anesthesia must be resorted to. Trichlorethylene on a sponge has been suggested in this situation. When the proptosis is due to an underlying lesion such as a tumor relief is obtained by treatment of the mass.

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SPECIAL ARTICLE

LETTER FROM INDONESIA

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BECAUSE most American physicians have not had the opportunity of visting Indonesia I should like to offer some information about my country, its population and its history in brief lines, some data regarding historical developments of what is called western medicine and concerning general conditions. It is believed that this information will help readers understand Indonesian problems in general and medical problems in particular.

The Republik Indonesia (Republic of Indonesia) was recognized as a sovereign and independent country as a result of the Round Table Conference at the Hague from August 23 to November 2, 1949, and was admitted to membership in the United Nations on August 14, 1950.

Several days after the surrender of Japan, on August 17, 1945, Soekarno and Hatta, respectively president and vice-president, proclaimed the Republik Indonesia on behalf of the Indonesian population; the first large battle between Indonesian Revolutionary forces against the occupying Allied forces took place on November 10, 1945, at Surabaya, and for five years no peace existed in Indonesia. Through the efforts of the United Nations Commission for Indonesia negotiations between the Republik Indonesia and the Kingdom of the Netherlands were begun, with the result mentioned above.

During the occupation by the Japanese forces (March 8, 1942, to September, 1945) Indonesians suffered greatly, as they also did during the years of revolution. Even at present there are disturbances of law and order in some parts of Indonesia: West Java, South Sulawesi (Celebes) and Atjeh. Approximately 40 per cent of the public expenses are used for the

*General practitioner in Djakarta.

armed forces, so that there is not much opportunity to conduct large-scale activities for development.

The Republik Indonesia comprises the former Netherlands East Indies, excluding Irian Barat (West New Guinea), which is still under dispute between Indonesia and the Netherlands. The Indonesian Archipelago is the largest in the world, covering approximately 735,000 square miles, with four main islands — namely, Java, including Madura (51,000 square miles), Sumatra (164,000 square miles), Kalimantan, formerly called Dutch Borneo (208,000 square miles) and Sulawesi, formerly called Celebes (73,000 square miles) — as well as some important smaller islands, such as Banka and Billiton, sources of tin, Bali and Lombok, well known as tourist centers, the Moluccas, where centuries ago Portuguese, Spaniards and Dutch were fighting each other for the monopoly of spices, and several thousand small ones.

This is not the first time in their history that Indonesians have had control over the Archipelago. In about the year 800 the Kingdom of Çriwidjaya — with the present Palembang, in South Sumatra, as capital — controlled the whole Indonesian Archipelago; so did Madjapahit in East Java with her famous prime minister, Gadjah Mada, who flourished from 1331 to 1364. Remains of Indonesians in Ceylon and in Visaya (Philippine Islands), as well as the famous temple at Borobudur, testify to the glory of Çriwidjaya under the dynasty of the Çailendras.

History

The Indonesian Archipelago is renowned for the fertility of its soil and its wealth in raw materials; it was known as such in the description of Chinese travelers in the second century concerning Yetiao (Java), Huang-Tsche (Atjeh), P'o-Li (Bali) and so forth. It was for several centuries a center of Hinduism, until at the beginning of the fifteenth century Islam made its entry into Indonesia. The oldest Hindu settlement was perhaps in 400 at Muara Kaman, at the border of the Mahakam River, in East Kalimantan (Borneo), according to the discovery of a stone inscription in the Sanscrit character (the oldest stone inscription in Arabic language is dated 1102).

The overthrow of Madjapahit (East Java) at the end of the fifteenth century by the Mohammedan King of Demak, in North Central Java, ended the Hindu period.

Western powers made their entry into Indonesia as follows: the Spaniards in 1521; the French in 1529; the Portuguese in 1536; the English (Francis Drake) in 1579; and the Dutch in 1594. (The Chinese had preceded the European powers in 1405.)

Under the mastery of the Netherlands the Indonesians were in revolt several times, the most severe of which was the one under the leadership of Dipanagara (Jogya) from 1825 to 1830. In 1908, the establishment of the national political party Boedi Oetama (Budi Utama) saw the beginning of an Indonesian-wide national movement.

The present Minister of Health, Dr. Lie Kiat Teng, is a member of the P.S.I.I. (national political party of Muslims), founded in 1912, just before World War I. No census has been taken since October 7, 1930. The Indonesian population is approximately 79,800,000, increasing yearly by 1.5 per cent, since the birth rate is about 35 and the death rate about 20 per 100,000 population. More than half the inhabitants live in densely populated Java; at present elaborate efforts are being conducted for large-scale transmigrations to other islands.

The Indonesians are mainly of the Malayan race. The original population is supposed to have been the Weddas, like the ethnologic groups living at present in isolated units as the Kubus, the Mamaks in the jungles of Sumatra and the Toalas in Sulawesi; the last resemble more or less the Senois of the Straits and similar groups in Cambodia and China. The aborigines retired to isolated areas on the invasion of the proto-Malayans in approximately 3000 B.C. Some groups in Gayo and Alas in Atjeh are supposed to be of the proto-Malayan origin.

The majority of the Indonesians are descendants of the deutero-Malayans from Indo-China, who came to Indonesia in about 300 to 200 B.C. The skeletons of the *Homo Pithecanthropus Erectus,* discovered by Eugene Dubois in 1890, and the *Homo Modjokertensis,* described by Dr. G. H. von Koeningswald, next to the discovery of skulls at Ngandong, which are like those of the Neanderthals, give an interesting chronology to this prehistory.

According to P. and F. Sarasin, the Indonesian Archipelago must in ancient times have been a part of South East Asia, inhabited by the Weddas, who are small and have dark complexions, like the people of Ceylon and other parts of Asia.

The lingua franca and official language in Indonesia is the *Bahasa Indonesia.* Besides the Bahasa Indonesia many languages are still spoken—Javanese, Sundanese, Madurese and so forth.

Social and Political Description

Religious liberty is granted to all who live in Indonesia. The majority—about 90 per cent—are Muslims; about 3 per cent are Christians, and the remainder are of other faiths. Indonesians are very tolerant in religious matters. A marriage between Muslim and Christian is not uncommon. The majority of the inhabitants of Bali belong to the Hindu religion, and traces of Hindu influence are still widely discernible in the Indonesian community. This attitude of tolerance is the basic reason why the majority of the people oppose the idea of making the Republik Indonesia an Islamic state as some groups in West Java, South Sulawesi and Atjeh in North Sumatra aim to do by means of force.

Republik Indonesia is democratic. Democratic institutions have existed in Indonesia from ancient times. The dessa, the smallest administrative unit of the country, is a living example of the democratic approach.

Economic Considerations

Indonesians are still in a bad position economically. Many large agrarian estates, covering approximately 2,500,000 hectares*—rubber, tea, coffee, sugar and cocoa—are owned and exploited by foreigners, who make great profits. The oil industry is entirely in foreign hands, as are other important industries. Foreigners have still dominant positions in trade, banking and traffic. It may be stated that there is still a colonial economy in the politically sovereign and independent Republik Indonesia.

One is ashamed to acknowledge that Indonesia must import almost everything for daily necessities, even rice, which is the main food. As in prewar days, Indonesia has to import 10 per cent of its need in rice. Rice production is now increasing, and it is hoped that imports of rice will no longer be necessary in three to five years; 600,000 tons were imported in 1952, and 350,000 tons in 1953.

Like all other Asiatic countries, Indonesia has nutrition problems, which are mainly a *protein problem*. Indonesians do not like milk and are not very fond of meat; huge quantities of dried and salted fish, which is a welcome dish in the Indonesian meals, are imported—about 60,000 tons yearly—in spite of the fact that the seas surrounding Indonesia are very rich in fish, equal to that of the seas of the whole of Western Europe. To solve the protein problem, Indonesians will have to reconquer the sea, as they conquered it centuries ago, in the time of Çriwidjaya and Madjapahit. Maritime activities ceased during the mastery of the Netherlands for about three and a half centuries.

According to one estimate the death rate from cirrhosis of the liver among male Indonesians above eighteen years of age is 4.29 per cent of the total death rate. This may be evidence of the close relation between a protein-poor diet and the occurrence of liver diseases.

In trying to improve economic and financial conditions and by doing so to improve living standards, the Indonesians have obtained much help from other countries and from the United Nations and its special agencies. However, the Indonesian foreign policy of active neutrality in the present tension between the Western Powers and the countries behind the Iron Curtain may cause some countries to discontinue help. This would be a great pity indeed since foreign aid has already had notable success in Indonesia, whose leaders are fully aware that it is but a part of the whole process of economic reconstruction and development of the country, in which the training of experts and the obtaining of capital goods are vital.

*The hectare is equivalent to about 2½ acres.

Medicine in Indonesia

With the arrival of Dr. Jacobus Bontius, author of *De Medica Indorum* (Leiden: 1642), Indonesia became acquainted with western medicine. This contact began in 1626.

An office for health care of the population (Burgerlijke Geneeskundige Dienst), established in 1827, was, until 1911, under one management, together with the military service. In 1851 a beginning was made with the training of Indonesian doctors, but not until 1927 was medical education placed on a university level by the establishment of a faculty of medicine at Djakarta. Ten years later there were, in all, only 542 medical students, of whom 100 were Europeans, 195 Chinese and 247 Indonesians, including 18 female students. At present there are 4 faculties of medicine in Indonesia: 1 in Djakarta, 1 in Surabaya, 1 in Jogyakarta and 1 in Medan (starting this year); still another will be established in Makassar next year.

In Djakarta alone the number of medical students at the end of 1952 was 1068—that is, 829 male and 239 female students. The yearly total of new students is about 250 for Djakarta, about 300 for Jogyakarta and about 250 for Surabaya. Lack of facilities and shortage of teachers are the main reasons why the number of newly admitted students cannot be increased. For entry into the medical faculty only a diploma of the S.M.A.-B is required. Intelligence and aptitude tests and interviews are not conducted. Before entering a university the young student spends six years at a primary school (sekolah rakjat) and three years at a secondary school (sekolah mengengah pertama) and another three years at the higher secondary school with the basic sciences as main curriculum (sekolah mengengah atas B). On entering the medical faculties students have an average age of eighteen or nineteen years.

The duration of the undergraduate medical training is seven years: one year in physics, chemistry and biology; two years in physiology, anatomy, pathology, parasitology, biochemistry, pharmacology, cytology, bacteriology, roentgenology and practical biochemistry; and two years in preclinical and another two years in clinical study, including thirty-seven weeks as coassistant, in the first year, twelve weeks in internal medicine, ten weeks in pediatrics, eight weeks in psychiatry and neurology, five weeks in the practice of pharmacy and two weeks in neurosurgery. In the second year of the clinical study he has to spend ten weeks in surgery, ten weeks in obstetrics and gynecology, four weeks in ophthalmology, six weeks in dermatology and venereology, three weeks in roentgenology, four weeks in otolaryngorhinology, three weeks in stomatology and three weeks in forensic medicine.

The aim of the medical curriculum in Indonesia is to produce a general practitioner with a sufficient basic knowledge and a sufficient skill to conduct a general practice immediately after leaving the medical faculty.

According to law, the young doctor has to serve three years in the Ministry of Health before he will get a license to practice.

At the end of 1939 there were 1277 doctors, 588 in governmental service and 689 with a private office or serving a nonofficial body. The yearly production of the two medical faculties does not exceed 60 doctors. At present there are about 1400 doctors, 300 of whom are foreigners, for a population of 79,800,000. Foreign doctors are coming more and more, on a three-year service contract with Republik Indonesia. Help expected from the United States includes the arrival, among others, of 10 professors from the University of California.

During the past three years the production of the medical faculties has not exceeded the prewar level. The same lack is expected in the next few years. After six to eight years approximately 300 to 400 students should be graduating each year.

Meanwhile, the training of auxiliary medical personnel has been increased and differentiated, and the Ministry of Education has appointed a committee on medical education to find ways of increasing the production of doctors by medical faculties to meet the increasing demands of the community on doctors and of changing the medical curriculum and the technics of teaching in accordance with the developmental trends of the Indonesian society.

In April and May, 1953, the WHO team of medical scientists, under the chairmanship of Professor Arvid Wallgren, of Stockholm, visited Indonesia. The team consisted of 13 members, including, among others, Dr. Joseph S. Barr, of Harvard School of Public Health, and Professor Ernest Boyce of the University of Michigan. So far as medical education is concerned, the activities of Dr. Edward Grzegorzewski, director of the WHO Division of Education and Training Services, and the views of the first World Conference on Medical Education are subjects of profound interest to the Indonesian medical society. The problem is that physicians are desperately needed, but on the other hand, medical leaders wish to maintain and to improve the present quality of undergraduate medical education.

The importation of too many foreign doctors would put too great a strain on the facilities of the country. Able young physicians have been sent abroad to be trained as future lecturers. For that purpose the opportunity afforded by the T.C.A. Colombo plan and others has been taken. It is wished that more could be sent.

Lecturers from foreign countries can be invited, but Indonesian students may not understand them fully, for their knowledge of foreign languages is not yet sufficient. The future generation will be in a much better position, because English is now compulsory in the secondary schools.

Doctors of every field in Indonesia are facing a tremendous task, especially those in public-health service, since facilities and funds are very limited, skilled auxiliary personnel scarce and the needs of the community excessive. The expenses of the Ministry of Health for 1953 were only 1.7 per cent of the total expenses of the country for the year. The importance of preventive and social medicine is significant in the health program. In comparison with those in other Asiatic countries hospitals are relatively good, but there are only about 9.5 beds for every 10,000 persons (7 for general purposes, and 2.5 for specific diseases, such as tuberculosis, leprosy and psychosis).

Conclusions

The problems of medical practice in Indonesia may be summed up by a description of his weekly practice at Djakarta by a general practitioner of eighteen years' experience, recently given in the *Madjalah Kedokteran Indonesia* (*Journal of the Indonesian Medical Association*, which is written mostly in the Bahasa Indonesian). It is a very incomplete picture, for frequency of infectious diseases at Djakarta is bound to seasons. The author complains of not having time to fulfill his task as a family doctor as most of his time is occupied by diagnostic and therapeutic procedures, which are only a part of his duty.

The week's practice included 535 patients seen, 748 consultations (30 on Sunday, 166 on Monday, 113 on Tuesday, 126 on Wednesday, 112 on Thursday, 111 on Friday and 90 on Saturday), and 4 deliveries.

Division of patients according to race showed 13 Arabs (8 males and 5 females), 15 Indians (7 males and 8 females), 48 Chinese (half of each sex) and 459 Indonesians (280 males and 179 females). One hundred and ten patients were under six years of age, 85 from six to twenty, 303 from twenty-one to fifty, and 37 from fifty-one to seventy-one.

The diseases treated included respiratory tuberculosis, syphilis, gonorrhea, typhoid fever, dysentery, erysipelas, measles, infectious hepatitis, malaria, helminthiasis, mumps, frambesia, trachoma, chicken pox, prostatic cancer, diabetes mellitus, vitamin deficiency, anemias, allergies, endocrinopathies, inflammatory diseases of the eye, otitis media, nervous disorders, arteriosclerosis, degenerative and other forms of cardiac disease, hypertension, nasopharyngitis, tonsillitis, influenza, lobar pneumonia, bronchopneumonia, bronchitis (chronic and acute), tonsillar hypertrophy, other respiratory diseases, diseases of the teeth, gastric and duodenal ulcer, gastritis, colitis and so forth.

MEDICAL PROGRESS

PROCTOLOGY (Concluded)

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BOSTON

BENIGN LYMPHOMA

Before 1939, when Hayes and Burr reported a single case of benign lymphoma of the rectum, they had been able to find previous reports of only 20 cases. In a more recent article they[20] mention 206 reported cases, to which they add another 22 cases of their own.

These tumors are usually single and occasionally multiple, and are generally within reach of the finger. Felt as freely movable submucous nodules, they vary from several millimeters to several centimeters in diameter and are usually sessile.

No symptoms could be ascribed to the lymphomas with certainty. They were accidental findings as a rule. Hayes and Burr observed 36 lymphomas in 22 patients. Treatment consisted of excision or fulguration, and the first case, reported in 1939, had shown no recurrence after twelve years.

Barba[21] described 2 cases of lymphoid hyperplasia of the rectum in children, 1 of which he considered entirely benign; the other he regarded as a transitional phase between benign and malignant lymphoma.

The first patient, a nine-year-old girl, had had a yellow mucoid discharge from the rectum. Several polypoid nodules were found just above the pectinate line, and a biopsy before admission had been reported as "lymphosarcoma." There were also several discrete anterior cervical lymph nodes, but a repeated biopsy was considered to show "benign lymphoid hyperplasia." The rectal lesions had disappeared a year later, without any treatment.

The second patient, a boy of eleven, had cervical adenopathy, hepatosplenomegaly and an enlarged abdomen. A little later polypoid masses were found in the rectum. A tentative diagnosis of Hodgkin's disease was made, and the cervical lymph nodes were treated with x-rays but without much effect. Five years later a laparotomy for crampy abdominal pain revealed striking enlargement of retroperitoneal lymph nodes, which had produced some obstruction of the sigmoid. Rectal examination thereafter disclosed multiple nodular lesions of the rectal wall, located 3 to 10 cm. above the anal outlet, and a barium-enema study showed obliteration of the normal mucosal pattern. Pathological examination of a rectal nodule in this case demonstrated benign lymphoid hyperplasia, and a cervical lymph node likewise revealed benign hyperplasia. X-ray therapy produced a 50 per cent improvement in the rectal situation. Four years later biopsies of the rectal nodules were reported as essentially the same, and the boy was asymptomatic, but the descending colon and sigmoid were thickened, nodular and not freely movable.

Barba believes that this case, though still benign, probably represents a transitional stage in the development of malignant lymphoma.

BARIUM GRANULOMA

Beddoe, Kay and Kaye[22] observed a submucous polypoid lesion 1 cm. in diameter on the posterior wall of the rectum 6 cm. above the anal orifice. The patient had had two barium enemas, without pain or other symptoms, four and six weeks before the nodule was found and excised. Pathological examination demonstrated intact mucosa on the surface of what proved to be a grayish-white nodule. This was microscopically a granulomatous lesion, with fibroblasts, histiocytes and multinucleated giant cells. Numerous refractile crystals were interspersed.

A spectrographic analysis of carefully prepared specimens revealed characteristic barium lines and thus established the diagnosis of a barium granuloma. Presumably, this had been caused by the submucous instillation of barium through an injury to the rectal wall in the course of one of the barium-enema examinations.

Although unlikely to be encountered very often such a case represents an unusual type of foreign-body granuloma and can serve as a warning to use increased care in the insertion of rectal tubes for x-ray examination or other purposes.

UNCOMMON MALIGNANT TUMORS OF THE RECTUM

Several uncommon malignant tumors of the rectum have recently been reported from several sources. They are of interest chiefly because of their rarity in comparison with ordinary adenocarcinomas, and the treatment is the same.

Cylindroma

Johnston, Dockerty and Dixon[23] describe a case of cylindroma (adenocarcinoma of the cylindroid type) of the rectum, treated by abdominoperineal resection. This type of adenocarcinoma usually occurs in the salivary and lacrimal glands, the mucosa of the nose and mouth, the trachea and bronchi and, rarely, the cervix and vulva. The authors were unable to find any previous report of a cylindroma of the rectum, which is a low-grade infiltrating tumor, with a tendency to repeated local recurrence and late metastasis.

The case was that of a forty-six-year-old man with

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a tumor in the lower rectum. It was thought at first to be an adenocarcinoma of the carcinoid type, and abdominoperineal resection in two stages, three weeks apart, was carried out. The growth, which was slightly ulcerated and 5 by 4 by 2.5 cm. in size, had extended through the bowel wall and invaded the prostate. The lymph nodes were not involved.

Carcinoid

Carcinoid, like the cylindroma, is a low-grade adenocarcinoma occasionally encountered in the rectum as a circumscribed, submucosal tumor, appearing yellow in cut section and not encapsulated.

Rosser[24] found 48 cases of carcinoids reported between 1941 and 1949, with only 5 cases located in the literature before that time, and he added 2 cases of his own. One of these patients died with extensive vaginal, lymph-node and liver metastases about six months after abdominoperineal resection. The other, subjected to a similar procedure, was apparently cured.

On reviewing the 48 cases previously reported, Rosser noted that there were single, submucous, yellow nodules in 28, multiple nodules in 2, polypoid nodules in 3, stenosing submucous tumors in 2, invasive tumors in 6 and metastases in 5.

Saxe[25] reports the case of a fifty-two-year-old Puerto Rican male with 3 separate carcinoid nodules in the rectum, about 1.5 cm., 1 cm., and 2 by 3 cm. in size. They appeared on inspection to be benign adenomas and were removed locally through a posterior approach, with incision and closure of the posterior rectal wall.

No follow-up data were given, and this type of procedure is not to be recommended.

Melanoma

Rosser[24] also described a malignant melanoma, in a man fifty-nine years of age, which had protruded from the anus and been replaced daily for four months. It lay just above a fibrous polyp and appeared as a black, ovoid tumor, apparently about 4 cm. in diameter. It was removed by abdominoperineal resection, and the patient was well for two years, after which lung and brain metastases led to his death.

Adenoacanthoma

Steinberg[26] reports a case of adenoacanthoma of the rectum in a forty-one-year-old woman. The symptoms were those of an ordinary early carcinoma, and examination revealed a firm, rounded tumor the size of a large grape on the left posterior wall about 10 cm. above the anus. Biopsy diagnosis was adenocarcinoma, but after removal of the tumor by abdominoperineal resection the microscopical diagnosis was reported to be epidermoid carcinoma; an adjacent lymph node showed epidermoid metastasis.

Several months later, there appeared to be granulation tissue around the colostomy that kept re-forming after cauterization. Biopsy was reported as showing "adenocarcinoma." The entire colostomy was then excised, and a new one created. At this time an exploration of the abdominal cavity disclosed no metastases, with no evidence of another primary tumor anywhere in the remainder of the colon.

Almost a year later a recurrent nodule in the perineum was found to be an undifferentiated carcinoma resembling the nonglandular component of an adenoacanthoma, and it showed no well differentiated adenocarcinoma.

The patient died nine months later, with evidence of liver involvement, but an autopsy was not obtained.

Paget's Disease of the Anus

Reports of only 7 cases of Paget's disease of the anus were found in a search of the literature by Pearson and McArt.[27] They described another case seen at the Indianapolis General Hospital in a sixty-eight-year-old Negro with bilateral inguinal-lymph-node enlargement. The perianal induration varied from 2 to 10 cm. in width and extended 2.5 cm. into the rectum above the mucocutaneous line. After a local excision of skin, with a pathological report of "carcinoma of the skin (epithelium and corium), associated with Paget's disease of the epidermis," and then a removal of one inguinal lymph node showing carcinoma, abdominoperineal resection, with dissection of the left iliac lymph nodes, was performed in two weeks. A month after this third operation a fourth procedure, consisting of dissection of the left and right inguinal and right iliac lymph nodes was carried out. The pathologist reported "Paget's disease of the skin and anus, and adenocarcinoma (highly differentiated) of the apocrine glands, with metastasis to one of four right inguinal nodes, the left inguinal being negative." The patient was dead a year later with generalized metastases proved by autopsy.

Cancer Complications

Beaconsfield[28] presents an interesting case of diffuse cellulitis of one leg in a thirty-five-year-old woman that was due to perforation of a carcinoma of the sigmoid into the iliacus muscle and extension to the leg along this muscle. This was thought at first to be a gas-bacillus infection of the leg, but without any obvious portal of entry; the correct diagnosis was made four days later at autopsy. Free incision of the leg, together with gas-bacillus antitoxin and penicillin, failed to rally the patient, who went rapidly into shock and coma and died in two days. The author mentions 2 similar cases, reported by Barrington and Gardham, that differed only in the fact that the perforations were due to diverticulitis rather than cancer.

I have personally observed this type of extension of infection from the pelvis into the leg in a patient with a high pelvirectal abscess and in another woman after extensive radiation for carcinoma of the cervix

with deep ulceration and infection laterally at the apex of the vagina.

A study of the urinary complications after excision of the rectum for cancer has been carried out by Watson and Williams[29] on 100 patients followed for a year. As a result of removal of the rectum there is, in the male, a backward displacement not only of the bladder but also of the prostate, a postprostatic recess thus being avoided. On cystoscopic study the internal meatus of the bladder is seen to be closed, and the internal sphincter forms a well defined ridge. In some cases a funnel neck definitely characteristic of nerve injury is seen. In women there is little or no displacement of the bladder.

Of 50 male patients 21 had no retention of urine (residua of 150 cc. or less within twenty-four hours of withdrawal of the catheter). The remaining 29 had some degree of retention, 16 having 150 cc. or over after the tenth postoperative day. On hospital discharge 44 had no significant retention of urine, 5 had 150 to 360 cc., and 1 had complete retention, requiring subsequent prostatectomy.

The authors, in a consideration of the causes of retention, do *not* find any kinking of the urethra. They believe that section of the levators, with dropping of the bladder and prostate, makes the angle of urination more nearly vertical in the recumbent patient and that urination is therefore more difficult. Of the 50 patients 29 were thought to have no prostatic enlargement, 12 slight, 7 moderate and 2 marked. Of the 9 patients in the last two groups, 7 had severe urinary retention, and yet only 1 of these had to have a prostatectomy done, the other 6 having their residual urine reduced to insignificant levels eight to twenty-one days after operation. Some degree of urinary infection occurred in 44 of the 50 cases.

This study should serve as encouragement to all who are engaged in the surgery of rectal cancer and are encountering such a high incidence of urinary retention in the postoperative period. It suggests that patience will avoid premature cystoscopy and transurethral resection.

Shaw and Green[30] report an interesting case in which ligation of the inferior mesenteric artery at the aorta, in association with abdominoperineal resection for carcinoma of the rectum, was followed by massive infarction of the entire large and small bowel except the first 70 cm. of the jejunum. An autopsy revealed that there had apparently been a gradual atheromatous occlusion of the superior mesenteric artery that had produced a collateral circulation from the inferior mesenteric. As a result, the ligation of the latter vessel at operation had shut off all circulation to the bowel normally supplied by the superior mesenteric artery, and gangrene inevitably developed.

Although this complication seldom occurs it is suggested that it can be avoided by a temporary occlusion of the inferior mesenteric artery to allow palpation of the mesentery above to be sure that pulsations are coming through from the superior mesenteric artery before the inferior mesenteric artery is ligated.

Ulcerative Colitis

At a symposium on ulcerative colitis before the American Proctologic Society, June, 1953, in which the participants were Chester M. Jones, Richard B. Cattell and Leland S. McKittrick, of Boston, and R. P Turnbull, of Cleveland, many points of interest and value were brought out.[31]

Jones has seen the onset of the disease in patients from four to over seventy years of age, with the majority in the late teens or early twenties, and about an equal sex distribution. Starting usually in the rectum, the disease occasionally extends into the ileum. Recurrences tend to be precipitated by unexpected and undesired pregnancy, emotional or physical trauma, intercurrent infections or gross dietary indiscretions, though the last are often tolerated if the other factors are not present. Jones mentions the wide variation in severity of the disease, the rarity of perforation, the seriousness of gross hemorrhage and the occurrence of perianal sepsis, fissures and fistulas as complications. Joint involvement occurs in about 10 per cent of cases and is not always, though often, cured by colectomy. Skin eruptions are common, and occasionally large, necrotizing lesions appear. Eye involvement may be serious. Occasional renal calculi and cirrhosis of the liver are seen, but only 1 liver abscess has been observed in Jones's thirty years of practice. Occasional endocarditis, septicemia and endocrine disturbances, such as amenorrhea, occur. Fluid balance is often a problem, especially after surgery. Jones believes that sulfonamides for fever are usually better than the antibiotics. He does not believe in rectal or colonic irrigations in this disease but emphasizes the value of an adequate, nutritious diet in treatment. Barbiturates are useful for relaxation. Patients often tolerate a planned pregnancy very well but are upset by unexpected and undesired pregnancy, as mentioned above.

There are two main indications for operation: to save life and to rehabilitate a chronically ill patient.

Cattell stressed the importance of medical and surgical consultations early in the disease, with combined supervision thereafter. At the Lahey Clinic about 50 per cent of the patients with ulcerative colitis are eventually operated on. He urged against undue delay in deciding on and performing ileostomy, avoidance of abdominal exploration because of the danger of peritonitis and avoidance of anal-canal surgery except simple drainage of abscesses. Appendectomy, in association with ileostomy, should never be done because of poor healing of the stump in the presence of the colitis. In the fulminating type of disease ileostomy is resorted to if medical measures have not caused definite improvement in five to seven days.

Future colectomy is planned whenever ileostomy

is performed. Of 189 patients in the past six years a third had combined ileostomy and colectomy in one stage, whereas two thirds were handled by a three-stage procedure—ileostomy, colectomy and abdomino-perineal resection. The multiple-stage procedure is definitely safer; the single operation is used only in patients in good condition.

From 1928 to 1952 817 patients with ulcerative colitis were treated at the Lahey Clinic. In these cases 858 operations were carried out on 413 patients.

Before 1946, in all operations on 194 patients, there was a mortality of 22.2 per cent, and 18 per cent for ileostomy alone, whereas in the period 1947-1952 the hospital mortality in a series of 219 patients was only 5 per cent. On the basis of a total of 545 operations on the 219 patients the operative mortality was only 2 per cent.

Of the 219 patients 11 died in the hospital, but 7 died subsequently at home of complications considered to be definitely due to the surgery rather than to the disease, thus raising the mortality from 5 per cent to 8.2 per cent. Since 1947 the mortality for ileostomy alone has been reduced to 4.3 per cent.

Turnbull has devoted much time and thought to the intelligent planning, execution and postoperative management of the ileostomy itself. For example, in the choice of a site for ileostomy on a patient, a Torbot ring is cemented to the skin at the spot of election on the right wall of the abdomen, for a twenty-four-hour period, at some time before operation, to determine if it lies easily in spite of leg motions and other movements of the body.

At operation the protruding ileum is wrapped loosely with three layers of vaseline gauze held together with skin clips and caught with sutures at the base to the tissues just under the skin and at the tip to the ileal edges. Then a polyethylene bag is applied to the skin for collection of the ileostomy contents, which may be carried into a bedside bottle if profuse. The gauze collar is slit after twenty-four hours because of swelling of the protruding ileum. The polyethylene bag is changed every two or three days, Karaya gum powder being used on the skin close to the stoma and within the circular disk of the bag, as a protective coating. The Torbot, Rutzen or Perma-type appliance replaces the bag after ten days.

Turnbull went into details about the early detection and treatment of ileal obstruction, which is a frequent complication and leads to rapid deterioration of the patient's general condition, owing to dehydration and electrolyte depletion. If this complication is not appreciated and dealt with by use of a tube to siphon off the contents of the ileum, and by general measures of replacement therapy, death may easily ensue. A "dysfunction tube" similar to the Foley catheter is used by Turnbull.

McKittrick, in summary, spoke of the localized proctitis seen in patients from time to time, which may remain about the same for years but in an occasional patient suddenly develops into a diffuse acute ulcerative colitis demanding surgery.

He spoke also of the ease with which a diagnosis of ulcerative colitis can and should be made by means of proctoscopic study in almost any patient even though he is quite ill. Thus, unnecessary and ill advised rectal operations can be avoided.

The value of early medical and surgical teamwork was further emphasized, together with a word of caution against relying too much on the psychiatric approach to treatment in a disease that is obviously a serious medical condition and must be so treated.

McKittrick does not believe that all patients subjected to ileostomy should necessarily have a later complete colectomy, particularly young men whose sexual functions would inevitably be seriously impaired by removal of the rectum. He thinks that a certain number of patients do very well with ileostomy alone, although he recognizes the necessity of colectomy in many cases and also the possibility of malignant degeneration in the diseased colon later on.

Counsell and Dukes,[32] in 63 cases of chronic ulcerative colitis studied at St. Mark's Hospital in London, found that cancer had developed in 11.1 per cent. Also, of 11 patients surviving the colitis for more than ten years, cancer developed in 5.

Rapid growth and early metastasis characterizes these cancers, and their physical characteristics make x-ray diagnosis difficult. There is often an intramural spread, with very little encroachment on the lumen of the bowel, — no recognizable surface tumor, — and the tumors are likely to be colloid or mucus secreting.

In the 13 cases in which cancer developed, 12 patients had metastases by the time a diagnosis was made.

Kleckner[33] reports figures based on 425 cases of ulcerative colitis seen in private practice from 1940 to 1950. He mentions the rare occurrence of diverticulitis with ulcerative colitis. Abscess and fistula occurred in 3 per cent of the series, carcinoma in 4 per cent, massive hemorrhage in 0.5 per cent and polyps in 9 per cent. Of the entire series operation was carried out in 25 to 30 per cent of the cases, the remainder being treated medically.

Volvulus

Hamlin[34] has studied the relative merits of reduction of volvulus of the sigmoid by intubation through a proctoscope, as compared with reduction by laparotomy. Bruusgard had reported, in 1947, the use of intubation 168 times, in 91 patients, in Sweden.

From 1935 to 1947, at the Massachusetts General Hospital, 22 operations were performed on 23 patients, the volvulus having been reduced in the remaining patient during a barium enema. Of the 22 patients cecostomy was performed in 11, an untwisting of the volvulus in 10, and resection for gangrene in 1. Four patients died — a mortality of 17 per cent.

From 1948 through 1951 11 cases of volvulus of the sigmoid were seen at this hospital. Three of these were reduced by barium enema under fluoroscopic observation, and 8 cases by intubation through the proctoscope. In this group, also, there were 1 immediate resection for gangrene and 4 later resections with anastomosis.

Hamlin offers a word of caution regarding the possibility of perforation with the proctoscope or tube unless care is taken, and he recommends the knee-chest position whenever possible.

This report is of great interest, for it means that, in a recent three-year period, volvulus of the sigmoid in all cases seen in a large general hospital, with 1 exception, was not regarded as an emergency surgical condition but was treated by an endoscopic maneuver with equal success. The 4 later resections with anastomosis were, of course, done to prevent the recurrence of the volvulus that is otherwise always a possibility.

Diverticulitis

Welch, Allen and Donaldson[35] reviewed 582 cases of diverticulitis treated at the Massachusetts General Hospital from 1942 to 1953. Sigmoid resection, with a mortality of only 2.6 per cent, was performed in 114 of this group, whereas in an earlier series (1926-1942), reported by Smithwick, only 33 of 333 patients had been so treated. This indicates a definite trend toward more radical management of the disease — a trend that is justified by the decreasing risk of resection, owing to improved technics, the use of antibiotics and the avoidance of serious complications, which would occur in a certain number of these patients if resection was not performed. The authors list the following positive indications for resection: repeated attacks of diverticulitis in spite of adequate medical supervision; patients under fifty years of age; persistent sigmoid deformity on x-ray study; and coincident urinary symptoms.

A single-stage resection, used in 40 of the 114 cases, is preferred under the most favorable circumstances, but a two-stage procedure was utilized in 6 and a three-stage procedure in 65 of the cases. Multiple-stage operations, with diversion of the fecal stream, are safer in the presence of perforation, fistula, obstruction and bleeding and in poor-risk patients from any cause. Resections averaging 20 cm. of bowel are advised, with mobilization of the splenic flexure whenever necessary to afford relief from tension.

This paper includes other valuable details of diagnosis and surgical technic, as well as an evaluation of other, less definitive surgical procedures for diverticulitis.

Megacolon

Swenson[36] discussed the differential diagnosis of chronic constipation and true Hirschsprung's disease in childhood. Children with constipation from faulty bowel habits and emotional factors — by far the largest group — must be differentiated from those with constipation of neurogenic origin as in Hirschsprung's disease — a relatively small group.

In the former group the difficulty starts in early life, at the age of two or three, and is not accompanied by any urinary disturbances and rarely by abdominal distention, and the rectum is found to be full of feces, with some incontinence.

In the neurogenic group, on the contrary, the constipation often starts in the first forty-eight hours of life and includes an atonic bladder and striking abdominal distention, with the rectum usually empty and no fecal incontinence.

A diagnosis of Hirschsprung's disease can be confirmed by careful barium-enema examination, a small amount of barium being used and oblique views with spot films being taken to demonstrate a narrow rectum and rectosigmoid, and dilatation of the bowel above.

Fistula

Anal fistula in infants is not common, and yet Venturo[37] has observed 7 patients between two and sixteen months of age in a five-year period during which 300 abscesses and fistulas were seen. These babies were all white, male infants, and in each case the fistula was lateral, rather than in one of the posterior quadrants as most commonly seen in adults.

The author cites Bacon, who reported that of 3100 cases of abscess and fistula only 39 were in infants, and Buie, who found among 1801 patients with fistulas only 9 in infants. Also, from the Jewish Hospital in Brooklyn, out of 115,000 pediatric admissions in thirty-five years, there were 2700 proctologic problems and 20 fistulas.

The 7 cases described by Venturo[37] were all simple fistulas and easily cured by incision and drainage of the sinus.

Books

From McGill University and the Surgical Staff of Royal Victoria Hospital, in 1952, under the editorship of H. F. Moseley,[38] there appeared a very good textbook of surgery with two chapters on the colon and rectum by H. S. Morton. These chapters, comprising about 30 pages, are well written and illustrated, with more colored plates than are usually found, and this is also true of the book as a whole. It is essentially a didactic work, covering the essentials of surgical diagnosis and treatment in concise and clear style.

McKittrick and Wheelock[39] recently wrote a small 94-page monograph on carcinoma of the colon as part of an *American Lecture Series* under the editorship of Dragstedt. This is a very readable and well illustrated discussion based primarily on McKittrick's extensive personal experience, and that of Daniel F. Jones before him, reinforced by references to the experience of their colleagues in Boston and elsewhere. The volume should be most useful to many surgeons.

An unusual book of 239 pages, written in 1951, by Grace, Wolf and Wolff,[40] of Cornell University, is devoted to experiments on 4 male patients with colostomies, under varying conditions of emotional stress and strain, the changes in appearance and activity of the exposed bowel being observed under carefully regulated conditions. Two patients had ulcerative colitis with an ileostomy and also with prolapse of the wall of the cecum. One of the 2 other patients had a transverse colostomy of normal bowel proximal to diverticulitis with narrowing, and the other a normal descending colostomy above a lymphopathia venereum of the rectum with stricture.

Observations were carried out with still and motion-picture color photography of the color, motility, mucosal fragility and estimations of the amount and quality of mucosal secretion, its pH and the concentration of lysozyme in the mucus of the colon, with a careful search for petechiae and ulcerations.

A thorough investigation of the past histories of the 4 patients, revealing their pattern of life, pleasures and frustrations and their upbringing and conflicts, made it possible to subject them experimentally to questions or remarks calculated to stir up a reaction to conscious conflicts in their minds, with resulting changes in the large bowel that could be observed.

This book is very interesting to read and should be of value to every physician, not only because it evaluates the much discussed psychogenic factors in ulcerative colitis but also because it traces the influence of these factors on the gastrointestinal activity of persons in normal health.

For those who like to carry out minor proctologic procedures in their offices attention might be called to a chapter written in 1952 on this subject.[41]

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MEDICAL INTELLIGENCE

ABUSE OF THE QUECKENSTEDT TEST

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ONE often hears that the Queckenstedt test was negative or positive and that unilateral jugular compression caused such and such a rise as compared to bilateral compression and so on. It seems that the fundamentals of the purpose of the test have been forgotten, and to reiterate them would not be amiss.

Bilateral compression of the jugular veins causes congestion of the venous return of the brain, which in turn is transmitted to the pacchionian villi, indirectly increasing the cerebrospinal-fluid pressure. For example, if the lumbar puncture needle is placed at the third lumbar interspace and there is a complete spinal-cord block due to a tumor at the twelfth dorsal segment, compression of the jugular veins will cause the cerebrospinal-fluid pressure to go up, but the increase will not be transmitted below the level of the tumor. The only practical value of this test consists in the demonstration of whether or not there is a complete subarachnoid block. Compression of the jugular veins, during lumbar puncture in a patient with headache, is never necessary and may bring on serious or even fatal complications.

If the patient has an intracranial tumor causing a rise in the spinal-fluid pressure, even if papilledema is not present (as in about 10 per cent of cases), and the jugular-compression test is done, the intracranial pressure can be increased enough to favor herniation of the cerebellar tonsils into the foramen magnum, with respiratory arrest.

Unilateral jugular compression was popular in the days of frequent mastoid infections with concomitant thrombosis of the lateral sinus. Today this condition is rarely met with; moreover, it is known that the numerous variations in the right and left lateral

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sinuses in the formation of the torcular Herophili seem to discredit the validity of this maneuver.

Conclusion

The most useful and perhaps the only indication for the jugular-compression test is to evaluate the degree of compression, if any, exerted by a lesion of the spinal cord or adjacent to it.

BY THE LONDON POST

The Practice of Medicine—Good General Practice—Too Many Afferent Impulses

John Lister, M.D.

London, England

THE British Medical Association has just held its annual meeting in Glasgow. Sir John McNee, who has only lately retired from the Regius Chair of the Practice of Medicine in the University of Glasgow, took an unusual definition of a professorial chair as the title of his presidential address: "The Practice of Medicine."

He recalled that Sir William Tennant Gairdner, a former occupant of the same chair, had been President in 1888 and had addressed the association on the subject, "The Physician As Naturalist." He had stressed the classical derivation of the word physician and acclaimed him a natural philosopher and essentially a student of nature. Sir John emphasized the fact that, in spite of the great advances since Gairdner's time, it is still essential for the physician to study the natural history of disease. In many cases a complete and accurate diagnosis can be reached by the simple observational methods of the trained naturalist, but in others it is necessary to proceed to further investigations. The old study of the natural history of disease and the new scientific methods must be combined in the modern practice of medicine. This has now become both complex and costly because of the increasing emphasis on the scientific approach. The growth of specialism and the development of research institutes have become inevitable. Sir John pointed out that a serious side effect of this trend is a lack of general physicians and surgeons to conduct undergraduate teaching. He said that men with a broad view were required to shape the course of medical training. He thought, indeed, that the family doctor is now the only nonspecialized member of the profession with a general but not expert knowledge of all branches of medicine. Because the majority of medical students eventually become general practitioners, perhaps the general practitioner should be brought more actively into teaching-hospital teams.

* * *

So much attention has been paid to the bad features of general practice in the Health Service that the Nuffield Provincial Hospital Trust promoted an inquiry into general practice at its best. For this inquiry Dr. Stephen Taylor investigated 30 practices, comprising 94 doctors selected for their professional reputation for good work. The practices varied widely in type, size and location. On the whole, the finest practice was found in middle-sized country towns, where the doctors are usually combined in partnership serving the town and surrounding countryside. The good doctor knows that the two enemies of good medicine are failure to examine the patient properly and failure to keep notes. He also appreciates the economy of employing ancillary help to relieve him of duties not requiring medical training.

Secretaries and nurses were employed by many of the doctors in the practices studied. The survey strongly suggests that successful group practice enables the doctors to "offer the public a better medical service than each could offer on his own." Group practice lends itself to activities like the sharing of diagnostic aids, closer association with dentists and the absorption into general practice of child-health work and physiotherapy services. Such developments are on the lines envisaged in the minds of those who planned that health centers should be the central features of general practice in the National Health Service. Financial restrictions have prevented any large-scale building of health centers by local authorities, and the Government has been quick to realize that it is far better for the doctors to develop group practice themselves. Indeed, the Minister of Health has now decided that a small annual sum of £100,000 shall be set aside for the encouragement of group practice. The money is to be used in the form of interest-free loans for the purpose of providing premises where a group of three to six doctors may work together.

Yet another word in praise of present-day general practice has come from the report of the Central Health Service Council, stating that there is little evidence that the doctor-patient relation has been affected adversely since the introduction of the National Health Service. Indeed, in some respects this relation is better — possibly owing to the absence of the money factor and to the increased co-operation between doctors.

* * *

An American professor of medicine told me that he had no television set in his house because he already received too many afferent impulses by day and he liked to reduce the number received in the evenings to a minimum in preparation for a refreshing night's sleep. Certainly, this is a day of afferent impulses, and it was perhaps not so inappropriate as might at first appear that the British Medical Association should open its annual scientific meeting with a discussion entitled "The Problem of Sleep." Neurologists, neurosurgeons and general practitioners

all contributed to the discussion and the *Times* summarized their observations for the lay public in a pleasantly frivolous leading article entitled "Poetry or Porridge."

One speaker mentioned the necessity of removing any stimuli that might prevent sleep but added that certain stimuli, such as those initiated by some after-dinner speakers and readers of scientific papers, might actually favor sleep! Although the point was made that sleeplessness is no great danger and that no certificate of death from insomnia has yet been filed, it may be recalled that the only sign of mental fatigue to which Mr. Gladstone paid any attention was the inability to sleep. The recipes for sleep are legion. Hypnosis has its place, and though this is frequently effected by the hypnotic drugs, there are other methods. The golfer may play shot after shot over many golf courses until he falls asleep from the sheer boredom of it all; another man may recite poem after poem, and George Borrow is said to have found that reading Sir Walter Scott was the ideal soporific. Finally, there are those who pin their faith on a nightcap, and there are even some who still believe in the effects of a good, hearty meal. Perhaps it was because of his Scottish heritage and because he was speaking in Glasgow that a Perthshire general practitioner assured the meeting that the best nightcap of all was "a plate of good, thick porridge." I doubt whether many south of the border will want to try this prescription, and although the specialist who urged his patient "to cast out mind-disturbing thoughts" is no doubt right, the paradox of trying to go to sleep remains. The harder one tries, the more wide awake one becomes.

CASE RECORDS
OF THE
MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

BENJAMIN CASTLEMAN, M.D., *Editor*

VIRGINIA W. TOWNE, *Assistant Editor*

CASE 40341

PRESENTATION OF CASE

A seventy-four-year-old man entered the hospital because of an injury of the right knee.

The night before admission, after a party, the patient fell down a flight of stairs and landed on the right knee. He was unable to get up and had to be carried upstairs by his friends. The next morning he was still unable to walk because of severe pain in the knee and was admitted to the hospital.

Four years before admission he had had a sudden episode of weakness and numbness of the left arm. During the past two years he had had slight dyspnea on ascending one flight of stairs. There was no history of orthopnea, edema, angina or "acute indigestion."

Physical examination revealed a clear, slightly emphysematous chest, a normal heart, good peripheral arterial pulsations, slight facial weakness on the left and a slight increase of deep reflexes in the left arm and the injured right knee, which was very tender and swollen, especially over the lateral condyle of the femur. There was no weakness of the left arm.

The temperature was 99.5°F., the pulse 84, and the respirations 20. The blood pressure was 140 systolic, 90 diastolic.

The urine, which had a specific gravity of 1.022, was normal. The hemoglobin was 13 gm. per 100 cc., and the white-cell count 14,800, with 66 per cent neutrophils, 22 per cent lymphocytes, 8 per cent monocytes and 4 per cent eosinophils. The platelets appeared normal. A roentgenogram of the right knee disclosed a linear, jagged fracture through the right patella, without significant displacement of fragments, and an oblique fracture of the femoral shaft extending from the posterior aspect anteriorly and entering the joint space through the lateral condyle at the intracondyloid groove. An electrocardiogram demonstrated a normal rhythm at a rate of 85, and a PR interval of 0.15 second; the Q waves were small in Lead 2, deep in Lead 3 and rather deep in Lead aVF; the T waves were upright in Leads 1, 2, aVF, V_1, V_2, V_3, V_4, V_5 and V_6 and low and upright in Lead aVL. The QRS complexes were normal in Leads V_1 through V_6. Electrocardiograms repeated five, eleven and fifteen days later showed no significant changes. The tracings were thought to be probably normal, although the possibility of an old posterior myocardial infarction was considered initially.

On the second hospital day 35 cc. of bloody fluid was aspirated from the right knee, and on the third day an open reduction of the fractured femur was performed. Postoperatively, the patient did well, and he was able to sit in a chair on the seventh hospital day. On the ninth day slight edema of the left leg was noticed, but there was no calf tenderness. On the morning of the tenth day while sitting up in a chair, he felt dizzy, spoke with slurred speech and was immediately put back to bed. He soon became asymptomatic, but during the evening, he complained of numbness of the left hand, and on elevation there was pallor and weak-

ness of the fingers. The next morning no pulse was palpable over the radial or brachial arteries or in the axilla; the hand could be extended freely, was somewhat cooler and was of normal color except on elevation. The heart and lungs remained unchanged. Anticoagulant therapy was given on the twelfth hospital day and was continued for a week. The prothrombin time on the thirteenth day was 19 seconds (normal, 13 seconds), and two days after the discontinuation of anticoagulant it was 15 seconds (normal, 13 seconds). The left hand returned to normal in four days. During this period he had a temperature of 100°F., and the white-cell count was 18,200.

He was doing well until the twenty-first hospital day, when he was found to be unresponsive in the morning. Examination revealed irregular pulse and respirations and unobtainable blood pressure; thirty minutes later, the blood pressure was 112 systolic, 64 diastolic, the pulse regular at a rate of 84, and the respirations also regular. He was sweating profusely and vomited frequently; the extremities were stiff, but the right side appeared more involved. The initial Babinski sign was positive bilaterally but later was elicited only on the right. Five hours later the left arm and leg moved spontaneously. About eleven hours after the onset he remained stuporous, with Cheyne–Stokes respirations, pupils that were 3 mm. in diameter and equal, normal eye grounds, supple neck and hemiplegia on the right. Lumbar puncture disclosed clear spinal fluid with a pressure equivalent to 122 mm. of water and a total protein of 28 mg. per 100 cc. He remained in a poor state, showing some responsiveness periodically. The Babinski sign on the right became negative ten days after the onset. The Cheyne–Stokes respirations continued, and hiccups developed. He had run a low-grade fever—temperatures between 99 and 101°F.—since the onset, but the lungs remained clear. A blood culture showed no growth, and the electrolytes were normal, but the white-cell count was 12,800. The urine gave a + test for albumin, and the sediment contained 2 to 35 white cells, occasional red cells and rare casts per high-power field. Gantrisin was given for the urinary infection. He improved slowly.

On the forty-first hospital day, a seizure involving the left arm and leg was noted. An electroencephalogram revealed diffuse abnormality, more severe on the right. The seizures were well controlled by Dilantin. After that he continued to improve slowly, became more alert, spoke occasionally under stress and moved both legs and the left arm. The temperature fell below 100°F. and then, on the fifty-seventh hospital day, rose to 101°F. He had some cough and regurgitation, but the lungs were still clear. The next night the temperature was 102°F., and there was some gastric retention. Moist rales were present at the base of the left lung; the white-cell count was 18,400. The temperature spiked to 104°F. the next day but returned to normal one day later; the lungs were again clear. However, during the day, tachypnea at a rate of 50 per minute developed, and he became unresponsive. The pulse was 110, the blood pressure 130 systolic, 80 diastolic, and the skin cool and dry. There were coarse rales in the lungs, especially at the left base. The abdomen was soft; the urine was clear. The next morning the blood pressure fell, the pulse became unobtainable, and the skin cold and moist, the temperature dropped to 97°F., and the extremities became completely flaccid. He died in the afternoon of the sixtieth hospital day.

Differential Diagnosis

Dr. Robert L. Berg*: From this protocol I can list four episodes: the sudden onset of weakness and numbness of the left arm four years before admission; the slurred speech, followed by numbness of the left hand; the unresponsiveness, sweating and vomiting; and the seizure. It would be helpful to know whether the sweating was on one side or the other, but it apparently was generalized. Did the patient have more than one attack, and was it a typical focal seizure?

Dr. John T. Quinby: The type of seizure was not too clear; it was probably twitching—at best it was not a focal episode. Over a period of a week he had several in which there was a fine, rapid, trembling motion of the left arm, shoulder and leg, with involvement of the left side of the face—not a discrete episode but one persisting over several minutes.

Dr. Berg: Was the initial attack similar to the others?

Dr. Quinby: Yes.

Dr. Berg: It was not a classic focal seizure?

Dr. Quinby: I should think not.

Dr. Berg: The patient probably died of some infection of the lungs or bronchopneumonia, but that was added to the other insults; it is the other insults that I am primarily going to discuss.

It is clear that this was a syndrome of multiple arterial obstructions, and the first differential point is to determine whether or not the obstruction was due to local disease in the arteries or whether it was disease elsewhere that had its effects peripherally. I shall be speaking of thrombotic disease of the various vessels involved under the heading of local disease and migrating clot or dissecting aneurysm under that of remote disease. There are general principles that are of some help in this regard. An obstruction from a local thrombosis is much more likely to be gradual in onset with something resembling an aura—if one can call it that—whereas obstruction from an embolus is more abrupt. With local thrombosis, therefore, there is probably much less peripheral spasm of the arteries supplied by the affected artery, with the result that recovery is less rapid and less extensive than after embolus or dissecting aneurysm, with which much more function is recovered in the next day or so as the spasm relaxes. I should like to discuss each of the

*Assistant physician, Massachusetts General Hospital.

four episodes from the point of view of these three diagnoses.

The first consideration is thrombosis. The first episode, which occurred four years before admission, is suggestive of thrombosis since it was not a very extreme attack, and still, after four years, there was a moderate residuum. In fact there apparently was a residuum in the face that the patient did not spontaneously comment upon; therefore, it was not enough to be concerned about or even noticed when it occurred, and yet it had persisted. I think that combination of events suggests thrombosis rather than embolus. After the second episode the slurring of speech could have been caused by a thrombosis; the arm was not as abruptly involved as it often is with embolus so that is not conclusive one way or the other. The third episode, in which he was found unresponsive, could have been due to a major thrombosis. The fourth episode, which I had believed to be a focal seizure, I thought could probably not be attributed to thrombosis, but in view of Dr. Quinby's comment it could be. I am left with the opinion that there could have been thromboses, but I am not enthusiastic about that possibility. Recovery, especially of the arm, was too rapid to be consistent with a thrombosis that developed as slowly as this condition did.

I might mention the types of thrombosis that this could have been. He had been operated on, so I think of the disorders that occasionally follow operation, such as fat embolus. Nothing suggested it particularly, and the period—ten days and twenty-one days—between those major episodes and the operation was long. There were no petechiae such as one might expect to see in the conjunctiva, skin or elsewhere with fat embolus. Postoperative thromboses, which Wright* relates to increased number and stickiness of platelets, also occur. Again, the period between the episodes and the operation was long; the platelet changes revert to normal by twenty-one days, Wright reports. Actually, brachial thromboses are quite uncommon compared to thromboses of the leg veins. If I decide that this was thrombotic disease, I shall settle on postoperative thrombotic disease as the type most likely to have been present.

This could have been an unusual giant-cell arteritis that involved the carotid and brachial arteries.

So far as dissecting aneurysm is concerned it would be remarkable for a patient to have one dissection followed in four years by another series of dissections. These too were separated in time—one was eleven days after the other—and selective in omitting the right arm along the way. That the second episode was coincident with the involvement of the brain and the arm is somewhat in favor of an aneurysm. It is difficult to attribute the findings to simultaneous thromboses in both areas at about the same moment, on the same day. That also presents a problem for embolization. The third and fourth episodes may have resulted from an aneurysm that, again, was selective. The progression and the direction of the development are consistent with an aneurysm that went from the right to the left artery in the head and then to the left brachial artery. There are two or three things against a dissecting aneurysm. A major dissection rarely occurs without pain and is almost always attended by heart murmur or by hypertension, neither of which this patient had.

The disease that I think he had is multiple emboli. With the exception of the first episode the illness could have been due to multiple emboli; however, the interval between the first and second episodes was so long that I am not ashamed to say that the first was a thrombosis and perhaps unrelated to the present illness. About the second episode, it is hard to explain why with emboli he had trouble in the left arm and in the brain simultaneously. I suppose that a sizeable clot may have been narrowed in the middle and parted company along the way. The third and fourth episodes are perfectly consistent. The seizure, which I now think of in terms of slight twitchings, may have been due to a number of conditions.

What could have been the origin of the multiple clots? He had symptoms of decompensation, as well as slight dyspnea for two years before admission, and he had electrocardiographic changes that were consistent with a posterior infarct. Because the changes did not evolve during hospitalization it is likely that this was an infarction of some age. It is possible that he had a mural thrombus overlying an old posterior myocardial infarction. There was no evidence of other sources of emboli such as murmurs to support rheumatic heart disease and subacute bacterial endocarditis or of a septal defect that could have transmitted a clot originating in the swollen left foot. I think, therefore, that it is most probable that he had an old posterior myocardial infarction and mural thrombus, with emboli to the left carotid and left brachial arteries and to the renal artery. It might be helpful to know why he fell downstairs. I suppose it is possible that that was part of the disease, but I do not believe it is necessary to postulate another cause for falling downstairs than his having been at a party.

DR. JOHN STOECKLE: When did you think he had the initial infarct?

DR. BERG: Some months before the fracture.

DR. BENJAMIN CASTLEMAN: And the emboli occurred later?

DR. BERG: Yes.

DR. CASTLEMAN: Ordinarily, emboli from a myocardial infarction appear earlier than several months after the original infarction. By that time the infarct has organized.

DR. BERG: Weeks, perhaps.

DR. JACOB LERMAN: I should like to ask if Dr. Berg would consider emboli from the venous side, such as

*Wright, H. P. Changes in adhesiveness of blood platelets following parturition and surgical operations. *J. Path. & Bact.* 54:461-468, 1942.

a thrombophlebitis of the legs, that reached the arterial side via a patent foramen ovale.

DR. BERG: That could occur, of course, and without a murmur, but it is more than I should like to swallow.

A PHYSICIAN: Would multiple pulmonary emboli explain the slight dyspnea?

DR. BERG: I think that is a good possibility, especially in view of the leg signs.

DR. STOECKLE: What caused the arrhythmia that was reported in the protocol?

DR. BERG: The record said that there was an arrhythmia, which disappeared fairly soon thereafter.

DR. CASTLEMAN: What is your opinion of the electrocardiogram, Dr. Wheeler?

DR. EDWIN O. WHEELER: Just what the record says; one cannot tell.

CLINICAL DIAGNOSES

Cerebrovascular accident.
?Embolism to left middle cerebral artery.
Fracture of lateral condyle of right femur.

DR. ROBERT L. BERG'S DIAGNOSES

Old right-sided cerebral thrombosis.
Old posterior myocardial infarction, with mural thrombus and emboli to left carotid, left brachial and ?renal arteries.

ANATOMICAL DIAGNOSES

Patent foramen ovale with paradoxical embolism.
Phlebothrombosis, right leg.
Cerebral infarcts, multiple, old and recent.
Superior-mesenteric-artery embolism, with early infarction of small intestines and ascending colon.
Pulmonary embolism, with early pulmonary infarction.
Bronchopneumonia.

PATHOLOGICAL DISCUSSION

DR. CASTLEMAN: Autopsy showed numerous foci of bronchopneumonia, with abscess formation as well as pulmonary emboli. There was a large embolus in the artery leading to the right lower lobe and small infarcts in the right middle and left lower lobes. Phlebothrombosis, probably as a result of the fracture and lying in bed, — the source of the emboli, — was present in the right leg.

DR. BERG: The left leg was swollen.

DR. CASTLEMAN: The thrombus was found in the right popliteal vein.

There was no dissecting aortic aneurysm, but there was a very long blood clot, 10 cm. in length and 8 mm. in diameter, lying free in the abdominal aorta (Fig. 1). It had the shape of an appendix and was only slightly adherent to the intima. It was obviously not locally formed and was characteristic of an embolus. The distal end of the clot protruded into the mouth of the superior mesenteric artery, and there was already some gangrene of the small bowel. The finding of a large patent foramen ovale and a definite groove in the midportion of the aortic embolus, very suggestive of the impression that could be produced by a valve in a large peripheral vein, makes this one of the very rare cases of paradoxical embolism. I believe this was the mechanism for the multiple arterial

FIGURE 1.

emboli — namely, from the leg veins to the right atrium, through the foramen ovale into the left side of the heart and out. The heart itself was normal in size, weighing 250 gm., with very little coronary-artery disease and no evidence of infarction.

DR. BERG: What did you see in the brachial artery?

DR. CASTLEMAN: The entire length of that artery was dissected, and we did not find anything.

DR. BERG: Perhaps an embolus just floated by.

DR. CASTLEMAN: The patient had had a recent cerebral attack. Dr. Richardson will discuss the findings in the brain.

DR. EDWARD P. RICHARDSON, JR.: There were two significant cerebral lesions, one of which was a large focus of old infarction in the right frontal lobe extending posteriorly to involve the rolandic cortex in the region of the arm-and-face area. Its appearance was consistent with a lesion of many years' duration. It was somewhat yellowish at the margins, suggesting that it had previously been a hemorrhagic infarct. This was undoubtedly the cause of the weakness and

numbness of the left arm four years before admission and of the minor neurologic signs — the facial weakness on the left and the spasticity in the left arm — that were observed initially. I also think that the left focal seizures that were observed later can be attributed to the cortical infarction in the right frontal and rolandic region and that one does not need to postulate a new cerebral lesion to account for them. The other lesion, which was much more recent, was an extensive infarct in the left cerebellar hemisphere and in the left half of the pons, supplied by a branch of the basilar artery. This lesion could well explain the sudden coma, with hemiplegia on the right, on the twenty-first hospital day. We are unable to give a clear explanation for the brief episode of slurred speech ten days after admission. It should be added that the cerebral arteries were free of atherosclerosis.

The yellowish appearance of the old infarction, suggesting that it had been hemorrhagic, is entirely consistent with what we have seen in proved cases of cerebral embolism. This, and the absence of atherosclerosis, make very likely the possibility that all the cerebral lesions were embolic.

DR. BERG: In view of the fact that you did not find occlusion of the brachial or of the left carotid artery would you consider that the long clot found in the abdominal aorta could have originated in the leg, gone through the heart and lodged temporarily at the entrance of the orifices of the left carotid and brachial arteries and then washed down into the abdominal aorta?

DR. CASTLEMAN: That is a possibility, but since the aortic embolus was apparently halted in its descent by entering the mouth of the superior mesenteric artery, the absence of abdominal pain and other signs of mesenteric occlusion indicate that this was a terminal event.

CASE 40342

PRESENTATION OF CASE

A thirty-five-year-old man entered the hospital because of lower thoracic and lumbar pain.

At the age of nine the patient had fallen from a horse and had pain in the lumbar spine for several weeks thereafter. He had been well for the subsequent eighteen years except for two or three short periods of pain after injuries to the back while he was playing football. Eight years before admission, when he lifted a heavy load, pain developed in the lower lumbar spine, shooting down the posterior aspect of the right thigh to the knee. There had been three or four flare-ups since then. Because the pain was aggravated by lifting of heavy objects he had avoided such exercise for the last two or three years. Four and a half months before admission, while reaching overhead, he doubled up with severe pain in the lower thoracic and upper lumbar region, to the right of the spine and radiating across the abdomen to the umbilicus. There was no pain on the left side. For the next four to six days, motion of the back, especially flexion, caused excruciating pain, which gradually subsided in about two weeks. Five days before admission the pain recurred, again radiating across the right side of the abdomen to the umbilicus, and he also began to feel tightness in the rectus-abdominis muscle on both sides. The pain was accentuated by motion of the spine, especially by twisting to the left. There was no leg pain, numbness or paresthesia. For the last three days he had been feverish and anorectic. He entered another hospital for study, and 10 cc. of procaine was injected to the right of the eleventh thoracic vertebra, without benefit. One day later neurologic examination revealed a guarded abdomen and absence of the cremasteric reflex on the right; the next day loss of the cremasteric reflex on the left was noted.

During the past year, he had also suffered several episodes of "gastroenteritis" with nausea, vomiting, anorexia, low-grade fever and nonproductive cough, that lasted for less than a week.

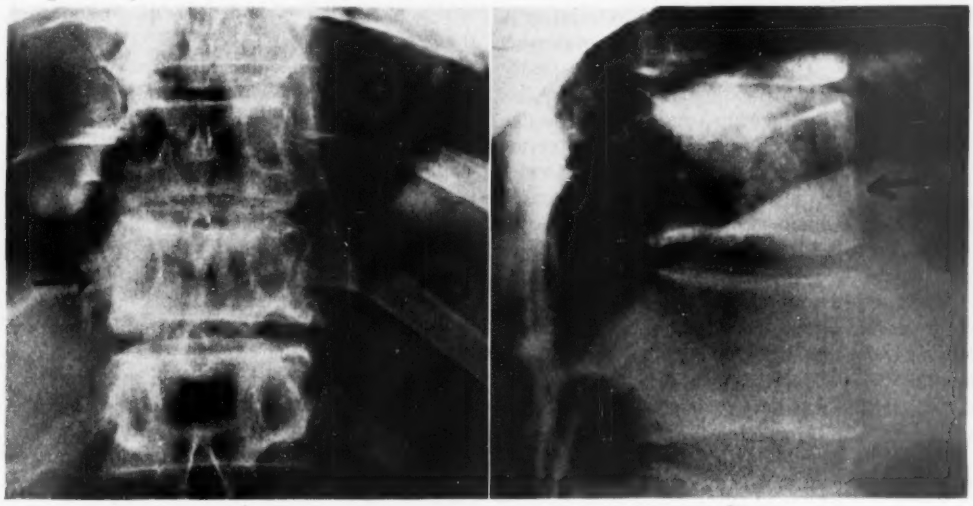
Physical examination revealed an acutely ill man who was unable to move because of severe pain in the lower thoracic and upper lumbar regions. The lungs were clear, and the heart normal; no abnormal masses were felt in the abdomen. There was fullness and exquisite tenderness in the right paravertebral region below the rib margin. Both motion and cough caused severe pain in that area. The lumbar lordosis was flattened. There was no tenderness over the spinous processes. Slight hyperesthesia was found at the inner aspect of the right upper thigh radiating from the penis downward for a distance of 8 cm. The deep tendon reflexes were active and equal. The abdominal reflexes were present in the upper quadrants and absent in the lower, the cremasteric reflexes absent bilaterally, and the plantar reflexes normal. There were no other sensory or motor disturbances. Straight-leg raising up to 70° on each side produced moderate pain in the back.

The temperature was 99.2°F., the pulse 95, and the respirations 20. The blood pressure was 114 systolic, 72 diastolic.

Urinalysis was negative. Repeated urinary examinations for Bence-Jones protein were negative. Examination of the blood revealed a hemoglobin of 13.5 gm. per 100 cc., a white-cell count of 13,600, with a normal differential, and an erythrocyte sedimentation rate of 43.5 mm. per hour. The white-cell count gradually returned to normal levels. The nonprotein nitrogen was 25 mg., the total protein 5.2 gm., the albumin 3.5 gm., and the globulin 1.7 gm. per 100 cc. The prothrombin time was 14 seconds (normal, 13 seconds). The sputum was negative for tubercle bacilli. Tuberculin tests were negative up to a strength of 1:100, and agglutination tests for typhoid and undulant fevers were also negative. A roentgenogram of the chest was normal. Multiple films of the spine re-

vealed definite bone destruction of the eleventh thoracic vertebra, and the intervertebral disk was narrowed between the tenth and eleventh vertebras; a questionable 8-cm. paravertebral soft-tissue mass was seen in the lower thoracic and upper lumbar regions, more marked on the left (Fig. 1).

Radicular pain along the course of the tenth and eleventh thoracic segments, absence of cremasteric and lower abdominal reflexes bilaterally and fleeting reflexes in the upper portion of the abdomen continued. On the eighth hospital day severe back pain followed coughing after a bronchial aspiration of sputum for examination. The next day there was a brief period of abdominal pain associated with decreased peristalsis. The back pain persisted, and two days later the patient complained of numbness at the left flank and midportion of the abdomen on the left associated with decrease of touch and pinprick sensations along the tenth thoracic segments. There was no urinary difficulty. The pain gradually lessened, but hypesthesia persisted. On the seventeenth hospital day, an operation was performed.



A B

FIGURE 1. *Anteroposterior* (A) *and Lateral* (B) *Views of the Lower Dorsal Spine, Showing Destructive Change and Partial Collapse of the Eleventh Dorsal Body and Minimal Fusiform Widening of the Paravertebral Soft Tissue at this Level.*

DIFFERENTIAL DIAGNOSIS

DR. THORNTON BROWN*: Did the patient lose any weight during the year before admission?

DR. JOSEPH S. BARR: I cannot answer that question; he was thin and looked as though he might have lost some weight. Does anyone know?

DR. VERNON JOHNSON: He thought he had lost approximately 10 pounds.

DR. BROWN: I assume that a determination of blood uric acid was done.

DR. BARR: It was within normal limits.

DR. BROWN: May I see the x-ray films now?

DR. JOHN F. GIBBONS: The films of the spine show destruction limited to the eleventh dorsal vertebra, and there is narrowing of the interspace between the tenth and eleventh dorsal vertebras. The original films, which show the rest of the dorsal and the lumbar spine, are not here. These spot films demonstrate the involved areas, and you will have to take my word for it that the changes are limited to this segment. In these projections, particularly in the lateral view, there is some downward buckling of the superior plate of the eleventh dorsal vertebra, and there is some diffuse abnormality in the trabecular pattern of the vertebral body. There is no localized osteolytic defect within the vertebral body, and the pedicles of the segment appear to be preserved. The line of pleural reflection at this level is widened bilaterally. The changes are limited to the vertebral body, and there is some destruction and collapse of the superior vertebral plate (Fig. 1).

DR. BROWN: Was there enlargement of the hilar lymph nodes or evidence of a primary tuberculous lesion?

DR. GIBBONS: No; the lung and heart shadow and the lymph nodes appear normal.

DR. BROWN: Was a skeletal survey done?

DR. GIBBONS: No.

DR. BROWN: The temperature was 99.5°F. on admission. Was the course in the hospital febrile?

DR. BENJAMIN CASTLEMAN: There was not very much fever.

DR. BROWN: This thirty-five-year-old man had had several episodes of pain in the lower back during his early life. Almost everybody has such a pain during life, and this man was engaged in heavy labor. He might have had some lesion in the lower back, but I do not think it had any bearing on his present problem. Four and a half months before admission, as the result of minor trauma,—namely, reaching upward,—

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he suddenly had severe pain of a radicular nature. If this had been a simple ligamentous strain or even a simple compression fracture one would have anticipated that in four months it would have more or less subsided. Therefore, he probably had some pre-existing lesion in the dorsal spine that resulted in collapse of the vertebra or a pathologic fracture that brought on the present symptoms.

He had had several episodes of gastroenteritis, with nausea, vomiting, anorexia, low-grade fever and a nonproductive cough. That certainly suggests some chronic illness; one would think of tuberculosis or a pulmonary neoplasm, but subsequent studies make this seem very unlikely. The fact that he had apparently lost some weight could be indicative of chronic disease. The physical findings are consistent with a destructive lesion at the tenth and eleventh dorsal vertebras producing root symptoms at that level—namely, radicular pain along the tenth and eleventh thoracic dermatomes. The lesion also probably involved the spinal canal, since he had lost the cremasteric reflex and had hyperesthesia over the first lumbar dermatome. There was therefore some lesion involving not only the tenth and eleventh roots in the intervertebral foramens but also the first lumbar and possibly the second lumbar roots within the canal. The first and second lumbar spinal segments, of course, are at this level.

He entered the hospital with a mild fever but apparently had not continued to be febrile. I am inclined to discount this fever because, although he had a slight elevation of the white-cell count on admission, he had a normal differential, and the white-cell count apparently returned to normal without specific therapy. The sedimentation rate was elevated. Was that checked?

DR. BARR: I think that a second test showed sedimentation rate to be about 20 mm. per hour.

DR. CASTLEMAN: He did have a mild fever; the temperature was around 99°F.

DR. BROWN: The remainder of the laboratory findings were normal. Of particular note is the normal or low-normal total protein and normal albumin-globulin ratio, which are against multiple myeloma. Apparently, fairly extensive studies were undertaken to rule out tuberculosis: the tuberculin test was negative in a strength of 1:100, and repeated sputum examinations were likewise negative. For this reason I think that tuberculosis is an unlikely diagnosis in this case. Similarly, the agglutination reactions for typhoid and undulant fevers were negative, and I shall therefore rule out the other more common types of osteomyelitis of the spine — namely, typhoid, paratyphoid and brucellosis.

Whatever this patient had tended to progress; for instance, after the coughing episode, the symptoms seemed to become somewhat more severe, and he apparently had a transitory paralytic ileus similar to that which follows compression fractures in acute injuries.

This destructive lesion, therefore, involved primarily the eleventh vertebral body, and the question is whether it was due to infection, neoplasm or one of the reticuloendothelioses. I think that tuberculosis can be ruled out on the basis of the laboratory findings. A low-grade osteomyelitis of typhoid, paratyphoid or brucella origin likewise seems extremely unlikely. It may have been osteomyelitis from one of the common pyogenic organisms, such as the staphylococcus or the streptococcus, but in four months one would probably have seen more soft-tissue reaction and more new-bone formation and sclerosis around the lesion than was present.

This leaves the neoplasms. This man, as far as I can tell, had not had any symptoms to suggest a metastatic lesion. Physical examination of the abdomen was negative, the chest film was normal, and the urine contained no blood. I do not think this was a benign tumor of the vertebra such as hemangioma or giant-cell tumor; the x-ray picture was not characteristic of either of those conditions. This leaves the primary malignant tumors.

Could this have been a multiple myeloma? I do not think so because of the normal laboratory findings: the total protein, the albumin-globulin ratio and the absence of Bence-Jones protein in the urine. It could have been a solitary myeloma, but this is not the characteristic x-ray picture; it is usually a single or loculated lesion and not a diffuse infiltrative process such as this. I do not believe there is any point in going over the other possible neoplasms—the rare ones such as chordoma. It could have been one of the reticuloendothelioses such as an eosinophilic granuloma or one of the other lipoidoses. This man was thirty-five; he was rather old for an eosinophilic granuloma, although this has been reported in the fourth decade. We do not have any other evidence for that. By exclusion I narrow the diagnosis down to lymphoma as the most likely primary lesion. In the last analysis, with a lesion like this, one is forced to resort to biopsy. I shall take as first choice one of the lymphoma group and as the second eosinophilic granuloma.

DR. WALTER BAUER: Has brucellosis been ruled out?

DR. BROWN: The patient had been chronically ill, which is suggestive of it, and he had a low-grade fever; however, the agglutinations were negative. I think, on the basis of that, I shall rule it out.

DR. RAYMOND D. ADAMS: I should think more along the lines of tuberculosis of the spine. There was definite evidence of involvement of two or three roots, which means that the process must have extended outside the bone for some distance. I am not certain that we have excluded tuberculosis though there is, as Dr. Brown has so lucidly explained, an equally good possibility of tumor. Certainly, exploration seemed justified.

DR. BROWN: You do not think it could have involved the tenth and eleventh roots laterally and

caused pressure on the first lumbar and second lumbar roots from midline extension?

DR. ADAMS: It must have involved two or more roots to give pronounced sensory loss.

DR. CASTLEMAN: Could that symptom result from a tumor extending out from the bone such as lymphoma?

DR. ADAMS: Yes.

DR. MARIAN W. ROPES: I should favor a cancer—possibly lymphoma—although I think tuberculosis cannot be entirely ruled out.

DR. CASTLEMAN: You do not think a negative tuberculin test in a strength of 1:100 rules out tuberculosis?

DR. ROPES: It very nearly rules it out, but I think no one laboratory test rules it out absolutely.

DR. CASTLEMAN: Dr. Barr, would you tell us about the opinion on the ward?

DR. BARR: The patient first came in on the neurosurgical ward with the presumptive diagnosis of epidural abscess. He had been seen in another hospital by one of the consultants in this hospital, who noted the absence of abdominal and cremasteric reflexes. He had an elevated white-cell count and fever and was acutely ill. When the destructive lesion of the eleventh thoracic vertebral body was seen on the x-ray films, he was transferred to the Orthopedic Service with a presumptive diagnosis of tuberculosis. Most of us who saw the original films thought there was a fusiform mediastinal shadow that extended over two or three vertebral bodies, and suspected that a paravertebral abscess was present. Subsequent events, I think, proved that that was not true. Tuberculin tests were run down to a strength of 1:100. With tuberculin that is well prepared, I have never seen a false-negative test at that strength in proved tuberculosis of the bone unless the patient is moribund from meningitis or some similar process. This patient was not moribund, and so we abandoned the diagnosis of tuberculosis and did a needle biopsy, inserting the needle into the vertebral body.

Dr. Castleman, would you like to describe that?

DR. CASTLEMAN: Unfortunately, we could not make very much out of the aspiration biopsy; it was inadequate; there were only a few cells.

DR. BAUER: Was it cultured?

DR. CASTLEMAN: So far as I know there was not enough tissue removed for both culture and section. The amount of tissue was inadequate for one to say anything about it so Dr. Barr did an open biopsy.

CLINICAL DIAGNOSIS

?Tuberculosis of eleventh thoracic vertebra.

DR. THORNTON BROWN'S DIAGNOSIS

?Lymphoma of the vertebral body.
?Eosinophilic granuloma of vertebral body.

ANATOMICAL DIAGNOSIS

Eosinophilic granuloma of eleventh dorsal vertebra.

PATHOLOGICAL DISCUSSION

DR. BARR: Through a costotransversectomy approach the body of the eleventh thoracic vertebra was exposed. We were expecting to enter a paravertebral mass, but there was no mass about the vertebral body, although there may have been some thickening of the retropleural tissues. The body itself, in its lateral aspect, was roughened and very thin, so that it was possible to go through its bony wall with a curet. We were able to scoop out material from the centrum extending from the tenth disk down to the eleventh disk without difficulty. In fact we removed the contents of the central portion of the eleventh thoracic vertebra. The appearance is very hard to describe; it did not look like tumor to me. There were bone trabeculae, which were soft, mixed with a little soft tissue that seemed to be of a fibrous nature. Dr. David Kahn made a frozen section and told me that it was tumor. I thought it was not infection and, having obtained enough material for a biopsy, closed the wound.

DR. CASTLEMAN: In the slide from the biopsy there were two types of cells: large, pale, reticulum-like cells and polymorphonuclear leukocytes with eosinophilic cytoplasm. This was therefore a characteristic eosinophilic granuloma of bone. The piece submitted for frozen section was made up primarily of large histiocytic cells, and it is understandable why they were interpreted as tumor cells.

DR. BARR: I might add a note about the course of this patient. After the frozen section he still had a great deal of pain; until the permanent microscopical sections came through we had supposed that this was a malignant tumor, perhaps lymphoma or Ewing's tumor, and we started x-ray therapy. Fortunately when we received the final diagnosis he had received exactly the right amount of irradiation for an eosinophilic granuloma, 500 or 600 r, so that the x-ray therapy was stopped at that point. We plan to do a fusion because he has a weak vertebra that will collapse if he gets up without it. The patient has done well since.

I think Dr. Brown should be congratulated for putting eosinophilic granuloma second. I had not thought of it, and I believe no one else had.

DR. CASTLEMAN: I believe the only cases of eosinophilic granuloma that have occurred in vertebras have been in patients with more than one lesion. It would be worth while to do a bone survey to see whether any other bones have been involved.

DR. BROWN: Vertebral involvement in children, with collapse of a single vertebra and without the demonstration of other lesions, has been reported.

DR. BARR: The so-called vertebra plana, or Calvé's disease, is due to eosinophilic granuloma.

ADDENDUM

A spinal fusion between the tenth and twelfth thoracic vertebras was performed, with a tibial graft, and the patient left the hospital two weeks later.

The New England Journal of Medicine

Official Organ of
THE MASSACHUSETTS MEDICAL SOCIETY
and
THE NEW HAMPSHIRE MEDICAL SOCIETY

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PUBLIC WELFARE AND THE PHYSICIAN

THE medical-care plan recently put into effect by order of the Commissioner of Public Welfare with the hope that it would meet with the approval of the physicians of Massachusetts was no doubt conceived with the laudable purpose of both saving money and achieving uniformity of procedure throughout the Commonwealth. It has been severely criticized on both theoretical and practical grounds.

To a good many people, the whole concept of public relief is theoretically unsound. They believe that the State may perhaps be entitled to regulate the social economy so that no able-bodied citizen is without opportunity to engage in gainful employment, but they are convinced that the Government has no obligation to provide direct aid in the form of money or services to those who are too old or infirm to work. According to this philosophy, such aid is a family obligation or possibly that of the local community. As a member of the local community the physician, of course, has a professional obligation to see that no one goes without necessary medical attention, whether or not the patient can pay for it. Neither the local community nor the State, however, has any right to *demand* such professional courtesy and compel the physician to treat anyone without recompense.

An alternative theory is that the Government owes the citizen the means of providing for himself certain necessities of life such as food, shelter, clothing and medical care. This is paternalism to be sure, but governmental paternalism has become increasingly common for many years, especially since politicians have discovered that a majority of citizens often prefer economic security to independence, and, repugnant as the idea may be to the almost extinct rugged individualist, paternalism is probably here to stay, at least until society goes bankrupt.

Under a paternalistic government the physician has no professional obligation to take care of poor people. The Government has assumed this obligation. The physician pays his share of taxes, and is no more obligated to work for nothing than any other citizen. To be sure, he then becomes a paid worker for the State and is a beneficiary of the paternalistic system along with the recipient of public welfare. He must therefore accept whatever recompense the State offers him, and he must work under its direction. Without any question, this is a form of state medicine. However, since a majority of citizens have not disavowed this degree of paternalism, the physician can only make the best of the situation.

The physician who objects to the whole idea of state-sponsored medical care has no relief except to attempt to arouse his fellow citizens and urge them to abandon the concept that the State has any obligation to provide charity to its citizens in any form.

Within the framework of paternalistic government, however, the only recourse of the physician who be-

lieves that he is being underpaid and overcontrolled by governmental officials is to protest vigorously, attempt to bargain with the State or, as a last resort, go on strike and refuse to work under conditions he considers unfair.

The theoretical solution is clear: abandon paternalistic government in all its various aspects. The practical solution is not so easy, since paternalism has been accepted by the majority and since the physician still feels the burden of his professional responsibility in the same way that the relatives of a pauper may feel an obligation to contribute to his support, even though he is subsisting at a low level on governmental charity.

ADMINISTRATION BILLS REJECTED

WHETHER in the long run for better or for worse, the physicians of the country, in company with the dentists, the veterinarians and other self-employed professionals, seem to have escaped inclusion in an expanded social-security coverage, even on an elective basis. This is as an organized, independent and self-reliant profession, skeptical of the advantages of the welfare state, wanted it to be. No one can honestly tell or clearly foresee what far-reaching effects a widening social-security coverage will have on the economic and spiritual future of a nation, nor can the present course of the wheel of progress be altered more than temporarily. Any doubts regarding the goal toward which it is turning will eventually be resolved on the basis of trial and error.

It is natural to envisage as a reward of human ingenuity and industry and as a result of national prosperity a more comfortable and a more secure existence for everyone than the majority of human beings currently enjoy. It is natural that the people who are governed should require their government to provide this security so far as it has the power to supply it. The terms on which it is made available, however, and the selection of the groups that should or should not be encouraged to enjoy it present difficulties beyond the capacity of the average person to decide.

The present administration may have been right or wrong in its attempt to extend old-age and survivor's insurance coverage to physicians, dentists and veterinarians, and these practitioners of their various arts of healing may have been right or wrong in bringing about its rejection. No fact is permanent, and the area that the welfare state will eventually include will be determined by the general will or acquiescence of its citizens rather than by the determination of any special group or groups.

The same philosophy applies to the bill that advocated the technic of reinsurance, designed, however inexpertly, to ease the financial burden of prolonged illness and stand as a bulwark against the evils of socialized medicine. Condemned by certain factions for its inadequacy and by organized medicine because it appears to this conservative group as another wedge in the split that the proponents of federal medicine are seeking to widen, it has been at least temporarily rejected by the present Congress.

This bill, a celebrated case in the eyes of organized medicine, is of minor importance when viewed politically. Its defeat, it is said, is a symbol of congressional independence; a price imposed by Congress for having enacted so much of the administration's proposed legislation.

As with rising waters that run into one channel after another, the swelling tide of social consciousness will not be stopped in its efforts to find a common level. The persistence of human aspiration in spite of failures, mistakes and misdirections is the encouraging fact of human existence. That which is right will eventually be accepted and will generally succeed; that which is wrong will be finally rejected.

RECEDING TIDE

THE wave of enthusiasm over the demonstration that cortisone greatly alleviates symptoms of rheumatoid arthritis and may initiate prolonged remissions in this disease was followed by a flood of uses of adrenocortical hormones in the treatment of a wide variety of inflammatory and noninflammatory diseases of varied or unknown etiology. The findings of many investigators who were exploring the various facets of these new physiologic agents were generally interpreted as encouraging their widespread and indiscriminate use in many diseases. But the enthusiastic reports were accompanied by strong reservations: the damaging effects of hyperadrenalism and the occurrence, activation or aggravation of severe latent infections suggested caution and a more reasoned evaluation of these agents.

Near the close of a panel discussion on endocrinology held at the April, 1953, meeting of the American College of Physicians,[1] the moderator found that all but three minutes of the hour and a half allotted to the discussion had already been taken up without mention of ACTH or cortisone. Dr. Edgar S. Gordon, who was called on to fill the gap, thought that he could cover the subject adequately in the remaining time. He made the following categorical statement:

> . . . the use of ACTH and cortisone in chronic diseases is becoming less and less justified, probably on the basis of the over-all, long time results, plus a great many complicating factors, including some which are quite dangerous. There are a good many of the best arthritis clinics in the country, for example, which have discarded cortisone therapy completely and have gone back to older methods. . . .

At a recent meeting of the Royal Society of Medicine, Dr. J. J. R. Duthie[2] expressed the view that there is no satisfactory evidence that rheumatoid

arthritis is due, directly or indirectly, to any abnormality in function of the pituitary-adrenal axis or to the production of abnormal steroid hormones by the adrenal gland itself. He believed that in most patients the natural course of the disease is not significantly altered by prolonged administration of either hormone and that it has become clear that the continuous administration of either of them in doses sufficient to achieve complete suppression of symptoms is "impractical because of the high incidence of side-effects." The presence of tuberculosis, dyspepsia, hypertension, renal disease, emotional instability, diabetes mellitus and osteoporosis were all considered contraindications to either cortisone or ACTH, and these conditions are most frequent in the type of patients who are likely to have rheumatoid arthritis. Duthie also called attention to the damage that may be done to the affected joints by excessive use, which suppresses the symptoms of tissue damage, and to the fact that "suppression of reactionary inflammation may, by allowing greater functional use, lead to an increase in damage of this nature." Even with the use of these agents to provide cover for orthopedic procedures in rheumatoid patients, the experience in Britain has been much less favorable than that reported from America, and he believed that the judgment of the value of hormone cover for operations and manipulations must be reversed. However, he stated that there was still room for their use in severe episodes for brief periods and also for the intra-articular administration of hydrocortisone in incapacitating involvement of a single large joint.

A detailed report of a carefully controlled experiment on a comparison of cortisone and aspirin in the treatment of early cases of rheumatoid arthritis carried out by a joint committee of the Medical Research Council and the Nuffield Foundation[3] deals with 30 patients given cortisone and 31 treated with aspirin. The dose was adjusted individually, and the patients were observed for a year. The course in these two groups was closely parallel in nearly all the reported characteristics — namely, joint tenderness, range of movement in the wrist, strength of the grip, tests of dexterity of hand and foot and clinical judgment of activity of the disease and of the patient's functional capacity. The only advantage observed was the somewhat more favorable hemoglobin levels and sedimentation rates in the cortisone-treated group; no other differences were noted. On each treatment the disease became inactive or only slightly active in about three quarters of the cases. About two fifths of the patients became capable of normal work or activity, and the therapies in these 61 patients offered surprisingly little choice.

An editorial note ends: "It would be ironical if the major practical outcome of the intensive work of the last few years is the discovery that aspirin, given in maximum tolerated doses, is the best and safest treatment for rheumatoid arthritis, the only common disorder for which cortisone is used."[4]

Obviously, the tide is receding, although perhaps somewhat more rapidly in Great Britain than in this country. It may now be possible more calmly to evaluate the role, however minor, of adrenocortical hormones in the management of rheumatoid arthritis and other conditions in which they have been used for purposes besides mere replacement therapy.

REFERENCES

1. *Panel Discussion on Endocrinology.* 20 pp. Summit, New Jersey: Ciba Pharmaceutical Products, 1954.
2. Duthie, J. J. R. Indications for ACTH and cortisone in rheumatoid arthritis. *Proc. Roy. Soc. Med.* **47**:323-325, 1954.
3. Joint Committee of Medical Research Council and Nuffield Foundation on Clinical Trials of Cortisone, ACTH, and Other Therapeutic Measures in Chronic Rheumatic Diseases. Comparison of cortisone and aspirin in treatment of early cases of rheumatoid arthritis. *Brit. M. J.* **1**:1223-1227, 1954.
4. Leading Article. Prolonged treatment with cortisone and ACTH. *Brit. M. J.* **1**:1249, 1954.

Our friend Prof. Rochester, some time since suggested to us that packing in the wet sheet might be a desirable remedy in cholera, and that the blood might thus receive again some portion of the water it had lost.

Boston M. & S. J., *August 30, 1854*

MASSACHUSETTS MEDICAL SOCIETY

COMMITTEE ON WELFARE PROBLEMS

A new medical-care plan prepared by Commissioner of Public Welfare Patrick A. Tompkins, effective July 1, 1954, establishes a uniform procedure throughout the State under which recipients of welfare assistance may receive medical care on terms prescribed by the Commissioner and his staff.

The Commissioner presented this plan to a committee of the Massachusetts Medical Society on January 1, 1954, with the announcement that it had resulted from prolonged negotiations with medical and ancillary professional associations and that it (or at least the fee schedules contained in the plan) was "binding" on their members.

A brief account of the development of the plan and its presentation for approval to the Society follows:

Early in the summer of 1952 the office of the Massachusetts Commissioner of Public Welfare communicated with Dr. John F. Conlin and with Dr. Thomas H. Lanman, at that time president of the Society, suggesting the desirability of reviving the Society's Committee on Tax-Supported Medical Care and proposed a meeting of representatives of the Society with representatives of the Department of Public Welfare for the purpose of discussing "the present unsatisfactory system" of fees for medical services to recipients of public welfare.

At the Council meeting of October 1, 1952, President Lanman reported that he was ready to appoint such a

committee. The committee subsequently constituted under the title of Welfare Fees Committee was as follows: Dr. Harvey A. Kelly, chairman; Dr. Fred Campbell; Dr. Philip Fortin; Dr. John E. Leary; and Dr. John F. Collins. Dr. Fred Campbell and Dr. John E. Leary were later supplanted by Dr. DeWitt S. Clark and Dr. Howard P. Kennedy. The office of the Commissioner of Public Welfare was notified of the composition of this committee on November 4, 1952.

On December 18, 1952, Drs. Kelly and Collins met with the Commissioner, Miss Vahey and Dr. Keenan at the Commissioner's office, at which time the Commissioner agreed to provide the committee with information concerning changes proposed in the administration of medical care for welfare recipients. No word, however, was received by the committee during the following six months.

On July 1, 1953, a meeting was held attended by the Commissioner and his staff and the full Committee on Welfare Fees, together with the officers of the Society, at 22 Fenway. This meeting was devoted almost entirely to the discussion of the proper fee level, and the committee succeeded in convincing the Commissioner that it should be raised. The difficulties concerned in establishing statewide fee rates were discussed. There was no mention of the fact that a complete new medical plan was contemplated.

At the Council meeting of October 7, 1953, the first report of the Committee on Welfare Fees was presented, and by vote of the Council the name for the committee was changed to Committee on Welfare Problems. There was still no inkling that an entirely new plan was in preparation by the Commissioner.

Following the meeting of July 1, 1953, the Commissioner's office was notified and it was well understood that any proposals from the Department of Public Welfare that were to be approved by the Society should be ready for consideration and in the hands of the Executive Committee before August 7, 1953, since the meeting of the Executive Committee was scheduled for September 9, 1953. No information was received; consequently the Council on October 7, 1953, was not in possession of any facts other than those contained in Dr. Kelly's report.

About the middle of December 1953 Dr. Kelly was notified that the Commissioner was publishing a new Medical Care Plan. The Commissioner's letter was dated December 9, 1953. He stated that he trusted that it would be possible for Dr. Kelly's committee and such other machinery of the Society as necessary, to concur in what he termed "these proposals."

The proposals referred to were: 1) a new fee schedule, 2) an agreement to appoint a committee for tax-supported medical care in each district of the Society, 3) information to be supplied as to the availability of medical services on an emergency basis, 4) publication of the principles and fee schedules in the New England Journal of Medicine.

The new plan was received during the week of December 27, 1953, accompanied by the announcement that it would go into effect on July 1, 1954. The Society was supplied with only twelve copies of the plan and local welfare boards received one copy each. These copies constituted the only source of information concerning the plan available to members of the Society.

A study of the provisions contained in this pamphlet of some 30 or more typewritten pages indicated that several of the provisions of the plan would be unacceptable to many members of the Society. Many councilors and well-informed members of the Society, including the members of the Committee on Welfare Problems, felt that the Commissioner had published the plan prematurely before sufficient opportunity had been given to the Society to study it. They still believe that it embodied a number of good principles and perhaps might be endorsed with certain reservations and changes. There were sporadic outbursts of violent criticism, however, from members of the Society who participated actively in welfare work, and it was obvious that a great deal of study would be necessary before the Society was in a position either to approve or disapprove of the Commissioner's plan as a whole.

On March 10, 1954, a second meeting was held between the Commissioner and his staff and the Committee on Welfare Problems with a number of invited representatives from various districts of the Society in attendance. At this meeting there was agreement as to the overall value of the new plan and that physicians should cooperate and assume responsibility in its execution. The four points of the Commissioner were accepted, with the exception that publication in the Journal would be accomplished only when and if the entire plan was completed and accepted. Furthermore, it was stated that any physician licensed by the Commonwealth should be eligible to participate. It was also brought to the Commissioner's attention that doctors recognized as specialists by the district committees should be designated specialists rather than board certified men only, and that the proposed medical consultant should be designated with the advice and consent of local committees only. Certain changes were also recommended in respect to reimbursing physicians for seeing more than one patient in a nursing home. One provision of the new plan provided that physicians attending welfare patients in community hospitals would no longer be paid for their services. The Commissioner refused to modify the plan in this regard. It was obvious after this meeting that there were a number of aspects of the plan which were entirely unacceptable to many fellows of the Society. Letters were received from various communities vigorously objecting to the plan either as a whole or in part.

The Executive Committee of the Council at its meeting of April 21, 1954, voted to recommend that the plan be neither approved nor disapproved and that action be taken only after it had been submitted and voted upon by each community in the entire state. This action was concurred in by the Council at the annual meeting of May 17, 1954 following which a comprehensive abstract of the plan was prepared by the Secretary of the Society. This was almost a verbatim transcript. It was mailed to each councilor and to each district secretary who was given five additional copies to distribute among those in his district whom he felt interested enough or qualified to vote approval or disapproval of each of the various sections of the plan.

On July 14, 1954, the Committee on Welfare Problems held a joint meeting with the Committee on Public Relations. There was general agreement concerning the most objectionable features of the plan, and at a subsequent meeting of the Committee on Welfare Problems with the Commissioner and his staff on August 4, 1954, these specific objections were presented to the Commissioner.

The Executive Committee of the Society and the Council at its October meeting will be given an opportunity to formulate the final reaction of the Society to the procedures outlined in the Medical Care Plan. In the meantime, fellows who wish to inform themselves concerning its content should obtain a copy of the plan either from the district secretaries, from the local welfare agency, or from the headquarters of the Society at 22 Fenway.

ROBERT W. BUCK, M.D.
Secretary

DEATHS

KOPPEL — William Koppel, M.D., of Newtonville, died on August 5. He was in his sixtieth year.

Dr. Koppel received his degree from Tufts College Medical School in 1919. He was a member of the staff of the Veterans Administration Hospital, Boston, and the New England Pediatric Society and, formerly, of the staffs of the Boston Floating, Boston City and Framingham Union hospitals.

He is survived by his widow, a daughter, a son and a sister.

ROUGHAN — Charles M. Roughan, M.D., of Lowell, died on July 11. He was in his seventy-fifth year.

Dr. Roughan received his degree from Tufts College Medical School in 1906. He was a member of the staff of St. John's Hospital and, formerly, president of the Middlesex North District Medical Society and school physician, Lowell.

He is survived by his widow, a daughter, two sons, three grandchildren and several nieces and nephews.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

CONSULTATION CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS

Orthopedic Clinic	Date	Clinic Consultant
Greenfield	September 2	Charles L. Sturdevant, M.D.
Gardner	September 7	Carter R. Rowe, M.D.
Haverhill	September 8	William T. Green, M.D.
Brockton	September 9	George W. Van Gorder, M.D.
Lowell	September 10	Albert H. Brewster, M.D.
Salem	September 13	Paul W. Hugenberger, M.D.
Lynn*	September 13	Daniel M. Killoran, M.D., and John J. Crowley, M.D.
Pittsfield	September 15	Edward J. Coughlin, Jr., M.D.
Worcester	September 17	John W. O'Meara, M.D.
Springfield	September 21	James D. Fisher, M.D.
Hyannis	September 23	Paul L. Norton, M.D.
Fall River	September 27	David S. Grice, M.D.

Rheumatic-Fever Clinic	Dates
Fitchburg	September 1, 8, 15, 22, 29
North Reading	September 14, 21, 28

Plastic Clinic	Held	Consultant
Eastern Massachusetts	Semi-monthly	Bradford Cannon, M.D.
Western Massachusetts	Monthly	Joseph M. Baker, M.D.

Physicians referring new patients to clinic should get in touch with the district health officer to make appointments. Patients are seen by appointment only.

*This clinic serves Lynn, Lynnfield, Saugus, Nahant, Revere, Chelsea, Everett and Winthrop. Referrals should be made directly to the Lynn Hospital.

CORRESPONDENCE

REGARDING INTERLINGUA

To the Editor: I was much interested in the editorial on Interlingua in the July 1 issue of the *Journal*. It seems clear that the use of *one* language in the world of science for translation and abstracting purposes would be of great advantage. In your very comprehensive editorial the impression is perhaps left that all, or many, of the projects mentioned are equally suited to serve in this role. But I think the choice of one such language over another can be greatly clarified if we separate the idea of "international language" into its natural constituents of "vocabulary" and "grammar."

The international vocabulary is already in existence and is in daily use in the great cultural languages of Western Europe. All modern international languages have used this vocabulary to some extent. The vocabulary of Interlingua, however, is unique in that it is based on many years of painstaking research in which the sole criterion for the acceptance of a given word was its internationality. Interlingua is therefore authentic since it is not invented but is rather the delineation of an already existing, living vocabulary. Of course, this vocabulary could be used with a variety of grammars, even including those of Esperanto, Ido, Novial and Occidental.

The vocabulary in itself, however, is capable of functioning as a complete language. The outstanding property of Interlingua is its immediate comprehensibility. If an Interlingua text were translated into English, French, Spanish, Italian, Portuguese, German or any other national language, its range of comprehensibility would be immediately restricted. If it were translated into Esperanto its range of comprehensibility would be even more restricted. This comprehensibility is therefore a tremendously valuable property of Interlingua that is not possessed by any other language, either natural or constructed.

Interlingua deserves the interest and support of scientific journals. It would be a great advantage to reintroduce this modernized and simplified form of Latin into the scientific world, where it could play the same secondary role to national languages as Latin did throughout the middle ages in scholastic circles.

I hope that we shall hear more of Interlingua in the *New England Journal of Medicine*.

John Lansbury, M.D.
Philadelphia

MEDICAL JOURNALS AND EQUIPMENT FOR TRAVANCORE

To the Editor: St. George's Mission Hospital is a nonprofit organization situated in a hilly village and working among the poor laboring classes of Punalur, Travancore, and its suburbs. Because good medical literature is very scarce in this part of the world, a small library was recently opened in the hospital with the idea of collecting used medical journals, books, bulletins, reprints of articles and transactions of medical societies from all available sources in foreign countries, so that up-to-date knowledge in medical practice could be obtained.

The Ayurvedic and Unani systems of medicine are very troublesome competitors to the allopathic system practiced here, and proper equipment and medical literature are essential for the successful management of the hospital.

It would therefore be greatly appreciated if readers of the *Journal* would send me back copies, of medical journals, medical books, reprints of articles and other useful medical literature and also second-hand surgical instruments, medical appliances, laboratory equipment and so forth, so that many poor patients may directly and indirectly benefit from them. This act of kindness will be gratefully remembered.

T. K. Thomas, *Medical Superintendent*
St. George's Mission Hospital
Punalur, Travancore
South India

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

The Yearbook of Psychoanalysis. Edited by Sandor Lorand, M.D. Vol. 9. 8°, cloth, 350 pp., with illustrations. New York: International Universities Press, Incorporated, 1954. $7.50.

This new volume in an important series contains 23 articles on subjects in psychoanalysis. The first article, by Siegfried and Suzanne Bernfeld, concerns "Freud's first year in practice, 1886-1887." The book is well published and should be in all collections on the subject.

The Adaptive Chin. By E. Lloyd DuBrul, M.S., D.D.S., assistant professor in charge of oral anatomy, College of Dentistry, University of Illinois, Chicago; and Harry Sicher, M.D., D.Sc., professor of anatomy and head of department, Loyola University School of Dentistry, and guest lecturer, Northwestern University Dental School, Chicago. 8°, cloth, 97 pp., with 47 illustrations. Springfield, Illinois: Charles C Thomas, 1953. $3.50. (Publication No. 180, *American Lecture Series.*)

The authors discuss the evolution of the human chin from fossil and natural specimens of the lagomorpha (rabbits and hares) through the primates. There is a bibliography of 90 titles.

Antibiotics Annual 1953-1954: Proceedings of the Symposium on Antibiotics. Chairman: Henry Welch, Ph.D., director, Division of Antibiotics, Food and Drug Administration. Oct. 28, 29, and 30, 1953. Washington, D. C.

Sponsored by United States Department of Health, Education and Welfare, Food and Drug Administration, Division of Antibiotics. In collaboration with the *Journal of Antibiotics & Chemotherapy*. 4°, cloth, 632 pp., with tables and illustrations. New York: Medical Encyclopedia, Incorporated. 1953. $8.00.

The papers read at this three-day conference cover the whole field of the subject. Short lists of references are appended to the numerous papers, and there is an index. The volume should be in all medical libraries and should prove of value to all persons interested in antibiotic therapy.

Mammalian Germ Cells. Edited for the Ciba Foundation by G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch. With the assistance of Margaret P. Cameron, M.A., A.B.L.S., and Jessie S. Freeman, M.B., B.S., D.P.H. 8°, cloth, 302 pp., with 54 illustrations. Boston: Little, Brown and Company, 1953. $6.75. *(A Ciba Foundation Symposium.)*

Many scientists participated in this symposium. The text is divided into two parts concerning spermatozoa and ova. Twenty-four papers were read and discussed. The printing was done in Great Britain, and the publishing is excellent.

Nash's Surgical Physiology. The first edition was written by the late Joseph Nash, M.D. The second edition was revised and edited by Brian Blades, M.D., professor of surgery, George Washington University School of Medicine. With the collaboration of Edward J. Beattie, Jr., M.D., Leon Gerber, M.D., Walter H. Gerwig, Jr., M.D., Ernest A. Gould, M.D., Alec Horwitz, M.D., Vincent M. Iovine, M.D., Gordon Letterman, M.D., William S. McCune, M.D., William C. Meloy, M.D., Hugo V. Rizzoli, M.D., and Jacob J. Weinstein, M.D., George Washington University School of Medicine. Second edition. 8°, cloth, 686 pp., with illustrations. Springfield, Illinois: Charles C Thomas, 1953. $12.50.

The first edition of this authoritative textbook was published in 1942. This new edition is the joint work of 12 members of the faculty of George Washington University School of Medicine. Some chapters have been completely rewritten, and others revised. Lists of references are appended to the chapters, and a bibliography of twenty-five pages concludes the text. There is an extensive index and the publishing is excellent. The type is especially good. The book is recommended for all medical libraries and to all interested persons.

An Introduction to Pathology and Bacteriology for Medical Students in the Tropics. By the late E. C. Smith, B.A., M.D., D.P.H., Sc.D. (Dubl.), M.R.C.P. Lond., M.R.C.P.I., D.T.M. & H. Eng. Revised by R. Kirk, O.B.E., B.Sc., M.D., D.P.H., F.R.F.P.S. Glas., M.R.C.P. Lond., F.R.S. Edin., professor of pathology, Kitchener School of Medicine, University College of Khartoum. Second edition. 8°, cloth, 390 pp., with illustrations. London: Staples Press Limited, 1953. $9.00.

The first edition of this special book was published in 1939. Dr. Kirk has revised the text of this second edition to bring it up to date. There is a good index. The printing was done in England, and the publishing is excellent.

The Roentgenologist in Court. By Samuel Wright Donaldson, M.D., F.A.C.R., roentgenologist, St. Joseph Mercy Hospital, Ann Arbor, Michigan. Second edition. 8°, cloth, 348 pp. Springfield, Illinois: Charles C Thomas, 1954. $7.75.

This valuable special book, first published in 1937, has been brought up to date. Although written for the roentgenologist it should prove useful to any physician or surgeon who has to appear in court. There are chapters on malpractice and its prevention, and considerable space is devoted to the novice witness — the person who has to appear in court for the first time or infrequently. Many illustrative decisions are inserted throughout the text. Short lists of references are appended to the chapters, and there is a good bibliography of 77 titles. There are also a key to citations, tables of cases arranged alphabetically and by state and an index. The book should be in the library of every physician and surgeon and in all medical libraries.

Disorders of the Blood. Diagnosis: Pathology: Treatment: Technique. By Sir Lionel E. H. Whitby, C.V.O., M.C., M.D. (Cantab.), F.R.C.P. Lond., D.P.H., Regis Professor of Physic, University of Cambridge, honorary physician to the United Cambridge Hospitals, honorary consulting hematologist to the Army, Ministry of Pensions and Ministry of Supply and lecturer emeritus in bacteriology to the Middlesex Hospital; and C. J. C. Britton, M.D. (New Zealand), D.P.H., physician and hematologist to the Prince of Wales's Hospital, London, consulting hematologist to Queen Mary's Hospital, Roehampton, and pathologist, St. Andrew's Hospital, Dollis Hill, Finchley Memorial and Hendon District hospitals. Seventh edition. 8°, cloth, 856 pp., with 20 plates and 106 text figures. New York: Grune and Stratton, 1953. $9.50.

The first edition of this standard British book was first published in 1935. This seventh edition has been extensively revised in certain portions of the text, and chapters on cytochemistry and biochemistry have been added. Lists of references are appended to the chapters, and there is a comprehensive index. The printing was done in Great Britain, and the publishing is excellent. The book should be in all collections on hematology.

German-English English-German Dictionary for Physicians in Two Volumes. By Prof. Dr. med. Dr. phil. Dr. med. dent. Fritz Lejeune, Vienna/Hamburg, and Werner E. Bunjes, lecturer in English at the Germersheim Interpreters' College of Mainz University. Vol. 2. English-German. 16°, cloth, 1737 pp. Stuttgart: Georg Thieme, 1953. $14.00. (Distributed by Grune and Stratton, New York.)

The first part of this new medical dictionary — German-English — was published in 1951 and has proved invaluable to persons interested in German medical literature. The book is now completed with this second part, English-German, which is recommended to all medical librarians and translators.

Michael Servetus: A translation of his geographical, medical and astrological writings with introductions and notes. By Charles D. O'Malley, Stanford University. 8°, cloth, 208 pp. Philadelphia: American Philosophical Society, 1953. $3.00.

This translation of the Latin works of Servetus into English is an important book for the medical historian and all persons interested in the history of medicine. The originals are scarce and expensive today, and the translations into a modern language are doubly welcome. Facsimile reproductions of the title pages of the original publications are given. The price is reasonable.

An Rh-Hr. Syllabus: The types and their applications. By Alexander S. Wiener, M.D., senior bacteriologist (serology), office of the chief medical examiner, New York City; assistant professor, Department of Forensic Medicine, New York University Postgraduate Medical School, and attending immunohematologist, Jewish Hospital of Brooklyn. 8°, cloth, 82 pp., with 20 tables and 8 figures. New York: Grune and Stratton, 1954. $3.75. *(Modern Medical Monographs* No. 9).

This small book presents the complete serology and genetics of the Rh-Hr types and gives brief directions for performing Rh-antibody tests and titrations by various methods. It also discusses erythroblastosis fetalis, exchange transfusion, hemolytic transfusion reactions, the racial distribution of the Rh types and their application in disputed paternity and autosensitization.

NOTICES

NATIONAL ASSOCIATION OF CLAIMANTS' COMPENSATION ATTORNEYS

The National Association of Claimants' Compensation Attorneys will hold its eighth annual convention at the Hotel Statler, Boston, August 28 through September 2. This will consist of a 6-day medicolegal program on personnel injuries. Especially interesting to physicians will be the medicolegal seminar on the afternoon of Wednesday, September 1, with Dr. Richard Ford, acting head of the Department of Legal Medicine, Harvard Medical School, and medical examiner of Suffolk County as moderator.

SELECTIVE SERVICE SYSTEM RECLASSIFICATION OF II-A REGISTRANTS

On May 4, 1954, the National Advisory Committee to the Selective Service System issued Information Release No. 15, pointing out that "every effort should be made to have those physicians in Priorities I and II and that portion of Priority III born after August 30, 1922, who will finish their internships and residencies by July first, apply at once for commissions so that there will not be a protracted period of waiting between the end of the hospital year and the call to active duty."

On November 10, 1953, Information Release No. 10, "Residents for the 1954-55 Hospital Year" was issued, pointing out that the hospitals should not appoint any physician in the groups above for further training.

These groups are most urgently needed to meet the future calls during the present fiscal year. Those physicians who do not apply for commissions should have their II-A classifications terminated.

Information Release No. 10 is as follows:

Priority I and Priority II.

Practically without exception these men should be considered available for service and should not be deferred to accept or to continue in residencies for another hospital year. If they desire to serve the hospital temporarily until such time as they are called, there is no objection, providing it is understood that they are not to be delayed beyond the time that they are called to service.

Priority III.

(a) *Those with double liability:* Those Priority III individuals who were under 28 years of age as of June 19, 1953 (now under 28½ years of age) have double liability and may be called at any time as regular registrants in addition to being subject to call as special registrants. They should enter service on the completion of their internship without exception. The Selective Service System has arranged to postpone the induction of such regular registrants, if the registrants apply for and receive a commission, until the armed forces can bring them to active duty in an orderly manner.

(b) *Those over 28 but under 31 as of June 19, 1953:* They are in the group that should supply the needs of the armed forces and should enter service as soon as they have completed their internship or current residency. If deferments are granted by the local board for this group of registrants and it becomes necessary for the armed forces to ask for a call for physicians, the Selective Service System may have to present physicians of Priority III who are over 31 and who are now actively engaged in the practice of medicine. It is highly desirable that it not be necessary to issue calls for Priority III men who have attained their 31st birthday.

The National Advisory Committee has not changed its position. It has repeatedly called attention to the fact that a physician's potential liability for military service should be considered in the appointment of residents. This applies also to the so-called pyramid system: persons selected for advancement to an additional year's service as they rise in the pyramid should be those who are not liable for military service.

It is essential that hospitals co-operate in this program so that the intent of Public Law 84 can be carried out and that physicians entering the services are from the group that should go first, according to the provisions of the law.

INDUSTRIAL HEALTH CONFERENCE

The seventh annual industrial health conference sponsored by the Chamber of Commerce, Houston, Texas, will be held in that city at the Shamrock Hotel, September 23-25.

Symposiums will be held on the following topics: "Industry in the Atomic Age," "Absenteeism, Alcoholism and Mental Health in Industry" and "Establishing Effective Health Programs in Industry."

FRAMINGHAM GENERAL HOSPITAL

Dr. Joseph Dorsey, of Boston, will speak on the topic "Trauma to the Nervous System" at the Framingham General Hospital on September 21, at 8:30 p.m. All physicians are invited.

SOCIETY MEETINGS AND CONFERENCES

AUGUST 28-SEPTEMBER 2. National Association of Claimants' Compensation Attorneys. Page 363.
SEPTEMBER, 1954-MAY, 1955. Venereal Disease Research Laboratory Courses. Page 285, issue of August 12.
SEPTEMBER 2-28. Consultation Clinics for Crippled Children in Massachusetts. Page 362.
SEPTEMBER 6-10. International Poliomyelitis Congress. Page 399, issue of March 4.
SEPTEMBER 6-11. University of Illinois College of Medicine. Annual Assembly in Otolaryngology. Page 1092, issue of June 24.
SEPTEMBER 6-11. American Congress of Physical Medicine and Rehabilitation. Page 246, issue of August 5.
SEPTEMBER 7-10. Nineteenth Annual Congress of the United States and Canadian Sections of the International College of Surgeons. Page 828, issue of November 12.
SEPTEMBER 12-15. Second International Congress of Cardiology. Washington, D. C.
SEPTEMBER 13-15. American Association of Blood Banks. Page 968, issue of June 3.
SEPTEMBER 16-18. American Heart Association. Annual Scientific Sessions. Washington, D. C.
SEPTEMBER 21. Framingham General Hospital. Notice above.
SEPTEMBER 22-24. Mississippi Valley Medical Society. Page 246, issue of August 5.
SEPTEMBER 22. New England Pediatric Society. Page 968, issue of June 3.
SEPTEMBER 23-25. Industrial Health Conference. Notice above.
SEPTEMBER 24. American Medical Writers' Association. Page 246, issue of August 5.
SEPTEMBER 30-OCTOBER 2. Canadian Society for the Study of Fertility. Page 285, issue of August 12.
OCTOBER 1 and DECEMBER 3, 1954; FEBRUARY 4 and APRIL 1, 1955. New England Society of Anesthesiologists. Page 1054, issue of June 17.
OCTOBER 7 and 8. Pan-Pacific Surgical Association. Page 1092, issue of June 24.
OCTOBER 8 and 9. Academy of Psychosomatic Medicine. Page 748, issue of April 29.
OCTOBER 11-15. American College of Physicians Course. Page 44, issue of July 1.
OCTOBER 16-22. American Occupational Therapy Association. Page 1092, issue of June 24.
OCTOBER 18-29. New York Academy of Medicine Graduate Fortnight. Page 44, issue of July 1.
OCTOBER 25-27. New England Postgraduate Assembly. Hotel Statler, Boston.
OCTOBER 25-30. Course in Care of Atomic Casualties. Page 285, issue of August 12.
OCTOBER 27. New England Obstetrical and Gynecological Society. Annual Meeting. Hotel Somerset, Boston.
OCTOBER 27-29. International Symposium on the Hypophyseal Growth Hormone. Page 1092, issue of June 24.
OCTOBER 31-NOVEMBER 4. American Institute of Dental Medicine. Page 968, issue of June 3.
NOVEMBER, 1954-MAY, 1955. Sanitary Engineering Center Training Programs. Page 122, issue of July 15.
NOVEMBER 3-5. National Society for Crippled Children and Adults. Page 44, issue of July 1.
NOVEMBER 12 and 13. Inter-Society Cytology Council. Page 968, issue of June 3.
NOVEMBER 15-26. Laboratory Diagnosis of Tuberculosis. (Course.) Page 246, issue of August 5.
DECEMBER 5. American Academy of Dental Medicine. Page 1092, issue of June 24.
DECEMBER 8. New England Pediatric Society. Page 968, issue of June 3.
DECEMBER 13-17. Congress of Obstetrics and Gynecology. Page 748, issue of April 29.
JANUARY 10-15, 1955. Course in Care of Atomic Casualties. Page 285, issue of August 12.
MARCH 7-12, 1955. Course in Care of Atomic Casualties. Page 285, issue of August 12.
MAY 4 and 5, 1955. American Psychosomatic Society. Page 286, issue of August 12.
JUNE 21-25, 1955. National Association for the Prevention of Tuberculosis. Page 540, issue of March 25.

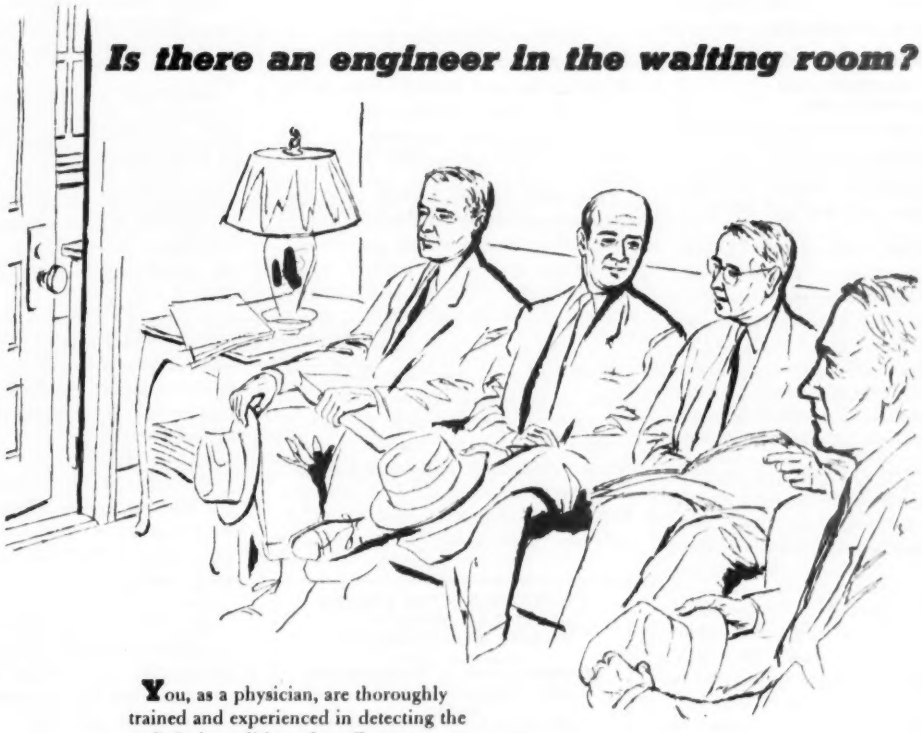
CALENDAR FOR THE WEEK BEGINNING THURSDAY, SEPTEMBER 2

THURSDAY, SEPTEMBER 2

*8:00-8:45 a. m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*9:00-10:00 a.m. Combined Medical-Surgical Rounds. Sherman Auditorium, Beth Israel Hospital.
*9:00-10:00 a.m. Arthritis Grand Rounds. Robert Breck Brigham Hospital.
*9:00-10:00 a.m. Cardiac Grand Rounds. Jimmy Fund Building. Children's Medical Center, 35 Binney Street.
*10:00-11:00 a.m. Medical Staff Conference. Sherman Auditorium, Beth Israel Hospital.
*10:00-11:00 a.m. Surgical Rounds. 5th floor teaching unit, Beth Israel Hospital.
10:30-11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

(Notices concluded on page xvi)

Is there an engineer in the waiting room?



You, as a physician, are thoroughly trained and experienced in detecting the clinical conditions that affect your patients' physical being. They depend on you completely for a knowledge and guidance not possessed by themselves. Conversely, do you not similarly look to professional men in other fields for aid when the need arises?

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If you are trying to decide which electrocardiograph to buy, we invite this type of comparison between the Viso-Cardiette and any other instrument. To make such an examination of the Viso possible, you may have a Viso for a 15-day trial* without any obligation whatsoever.

* This EXCLUSIVE plan places a Viso-Cardiette in your hands for 15 days. At the end of that trial period, if you are not completely satisfied with the instrument, you simply return it to us and that is all! You're under NO OBLIGATION.

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PHYSICIANS' Office FURNITURE. All standard lines, new. Used equipment from time to time. Convenient terms. Visit our showroom close by Veterans Hospital. Ample parking. T. J. NOONAN Co., 408 South Huntington Avenue, Jamaica Plain (Boston 30), Mass. Phone JAmaica 2-8700 for literature. Medical hospital, laboratory, sickroom supplies. B260-20-tf

NISSEN'S INSTITUTE OF PHYSICAL THERAPY, 1126 Boylston Street, Boston, KE 6-1030. Cape Cod office, Route 28, West Harwich. Telephone Harwich 984. B290-3-tf

CAMDEN, MAINE, "Undercliffe." Guest house, spacious rooms, also cabin. Two-room kitchenette, bath. Half mile to beach. Rates upon request. Mrs. Harry F. Melching, Camden, Me., Box 233. Camden 2796. B94-1-9t

FOR SALE—Ideal deluxe whitestone 5-story building. Auto parking. Facing Charles River. Heart of medical center. $65,000. Charles Worden, Sr., 298 Beacon Street. Boston. Shown by appointment. CI 7-8000. B91-25-tf

4-ROOM DOCTOR'S OFFICE completely equipped, in home. Air conditioned. Separate entrance. Excellent location, in West Medford. Address A37, *New Eng. J. Med.* 14-tf

BIND your *New England Journal of Medicine*. Make it a permanent part of your library—for easy reference and lasting wear. In full black library buckram, only $3.30 per volume, plus shipping charges. All other journals and periodicals bound also. Inquiries invited. Norden Bindery, 2100 West Grand Avenue, Chicago 12, Ill. B300-14-tf

RADIOLOGIST, Board Certified, seeks part-time association in hospital or office practice in Boston or vicinity. Also available for locum tenens and weekend coverage. Address A95, *New Eng. J. Med.* 8-3t

X-RAY UNIT for sale at half price, (General Electric—1951) for office. Used very little. Address A115, *New Eng. J. Med.* 9-2t

LEARY LABORATORY
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Timothy Leary, M.D., *Director*
Dependable and Rapid Laboratory Service. All Branches
Containers furnished
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FOR SALE—Ideal location for professional man for home and office. Deluxe suite facing Charles River, plus income. Furnished. In heart of medical center. Charles Worden, Sr., 298 Beacon Street, Boston. Shown by appointment. CI 7-8000. B90-25-tf

FOR SALE — Combined home and office on prominent corner in the Newtons. Excellent arrangement for general practice or specialty. Suitable for two offices. Additional apartment available. Property in excellent physical condition. For further information, call BI 4-3427, evenings, Sundays, LA 7-5506. B111-6-4t

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OFFICE FURNITURE—New—Used—Refinished. Bought and sold. Desk Clearing House, 115 Broad Street, Boston. HU 2-1318. B72-22-tf

UNITED LIMB & BRACE CO., Inc., manufacturers of artificial limbs. 61 Hanover Street, Boston, CA 7-2183. B517-6-tf

WANTED—Skin specialist and allergist for new clinic in Framingham area. Phone Mr. Harold Reed, TRinity 3-8800. B97-2-8t

WELLESLEY — Colonial home suitable for doctor, or for 2-family investment. Fine condition, excellent location. WEllesley 5-4087. B99-2-8t

DESIRABLE LOCATION, center of medical district, 15 Bay State Road. Entire basement for rent for medical offices or laboratory. KE 6-2270. B117-8-tf

WANTED—Monthly accounts, preferably to be done at my home. Address A109, *New Eng. J. Med.* 8-2t

BAR HARBOR, MAINE—For Sale—Doctor's home and offices, consisting of waiting room, examining room, treatment room, large laboratory and storeroom. Has been in use as doctor's home for 35 years. Address A114, *New Eng. J. Med.* 9-6t

STORROW HOUSE
(Former Storrow Estate at Lincoln, Mass.)
Staffed and Operated by the
MASSACHUSETTS GENERAL HOSPITAL
For nonpsychiatric ambulatory and semi-ambulatory patients needing rest or convalescent facilities.
CHARGES: $7.00 to $10.00 per day
Transportation for visits to physician's office provided. For information, call LIncoln 6-0343 or Massachusetts General Hospital Admitting Office LA 3-8200.

IF YOU ARE INTERESTED IN ORGANIZING a medical group practice in Dorchester, call evenings, after 6 p.m., BI 4-0214 or address A102, *New Eng J. Med.* 4-tf

ATTENTION DOCTORS — Registered physical therapist will treat your patients at homes, hospitals, convalescent homes. Excellent references. Anna T. Curran. CO 5-8896. B119-9-3t

OPENING for 1 resident at the Gaston Hospital, Dallas, Texas. Approved by American Board for 1 year general surgery. Salary $200.00 per month and complete maintenance. B30-11-tf

WANTED — General practitioners or internists to practice in suburb of Boston, 18-20 miles, west. Phone TRinity 3-8800. Part-time position — stipend — $3,000 to $5,000 per year. B109-5-8t

GENERAL PRACTITIONER AND ANESTHETIST wanted to work with surgeon, eastern Kentucky. Interview in Boston. Address A111, *New Eng. J. Med.* 8-3t

RADIOLOGIST—Certified in radiology 1951, 34, married, children, desires association with hospital or group in New England. Address A112, *New Eng. J. Med.* 8-3t

GENERAL PRACTICE available in town 20 miles from Boston. Prosperous community. Furnished office available. R. S. Whitney, 41 Church Street, Holliston, Mass. Holliston 2425. B114-7-tf

WANTED — Physician to take over practice in busy ski resort. Will rent with option to buy, fully equipped office including x-ray and 3-bedroom home with new kitchen, panelled living room with fireplace. Box 57, Stowe, Vermont. B118-8-2t

EXPERIENCED MEDICAL SECRETARY seeking position, New England area preferred. Telephone HA 6-2716, after 6:00 p.m., or address A113, *New Eng. J. Med.* 9-1t

(Advertisements continued on page xvi)

Thephorin 'Roche'

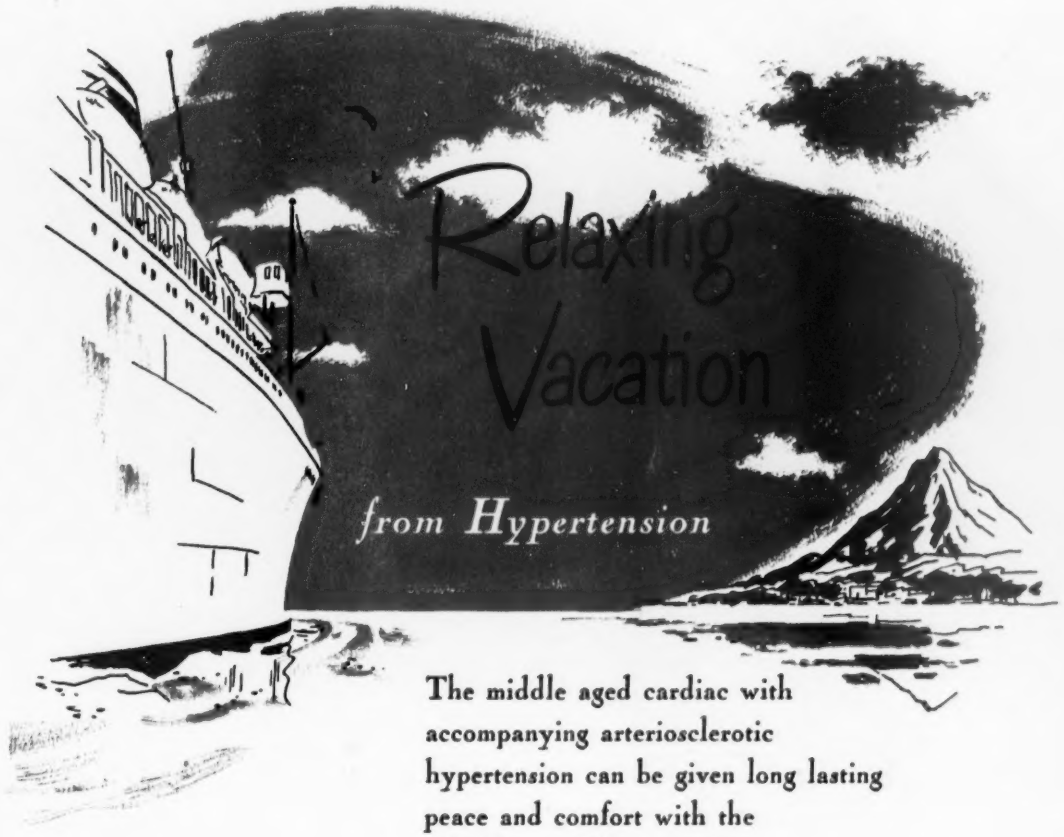
the daytime

antihistamine —

Thephorin is a potent antihistamine basically different in structure from all other antihistamines -- different also in its action -- it usually relieves allergic symptoms without drowsiness. Out of more than 2000 patients treated with Thephorin, 97% were alert and wide awake during therapy.

About Thephorin therapy in hay fever—

Over 79% of 859 patients suffering from hay fever were relieved by Thephorin.® This daytime antihistamine usually provides convenient control of allergic symptoms without drowsiness. Available in 10-mg and 25-mg tablets, plus anise-flavored syrup.



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yellow tablets, each containing theobromine calcium gluconate 500 mg. and phenobarbital 16 mg.

For diuresis and myocardial stimulation

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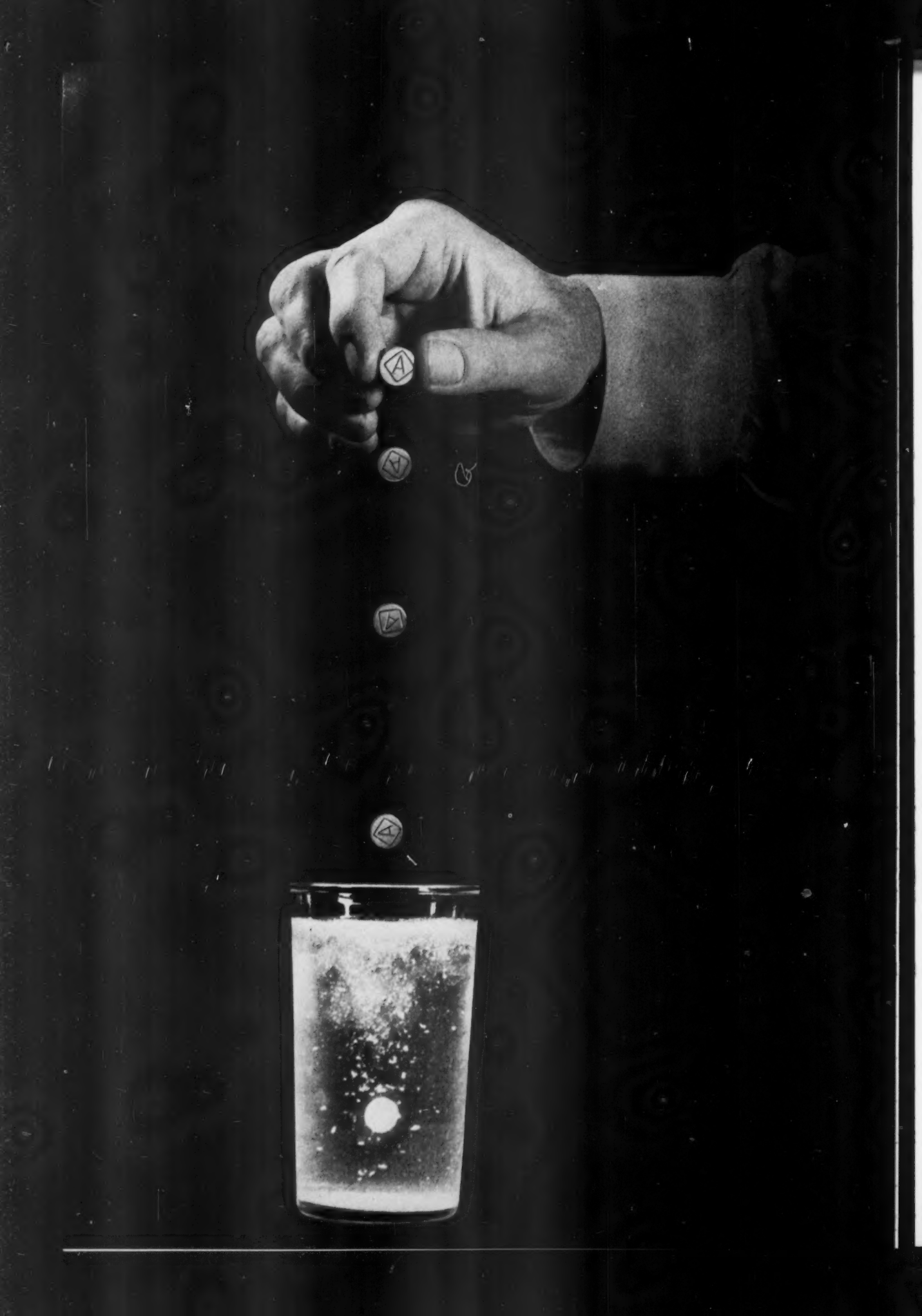

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starts to dissolve

NEW **filmtab* ...for faster drug absorption**

Now, *there's no delayed action from an enteric coating.* The new tissue-thin *Filmtab* coating (marketed only by Abbott) starts to disintegrate within 30 seconds after your patient swallows it—makes the antibiotic available for immediate absorption.

NEW **filmtab* ...for earlier blood levels**

Because of the swift absorption, your patient gets high blood levels of ERYTHROCIN (Erythromycin Stearate, Abbott) in *less than 2 hours*—instead of 4-6 hours as before. Peak concentration is reached within 4 hours, with significant concentrations lasting for 8 hours.

NEW **filmtab* ...for your patients**

It's easy on them. Compared with most other widely-used antibiotics, *Filmtab* ERYTHROCIN is *less likely to alter normal intestinal flora.* Prescribe *Filmtab* ERYTHROCIN for all susceptible coccic infections—especially when the organism is resistant to other antibiotics. Bottles of 25 and 100 (100 and 200 mg.). Abbott

**TM for Abbott's film sealed tablets, pat. applied for*

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Index to Advertisers

	PAGE
Abbott Laboratories	xiv, xv
The Armour Laboratories	vi
Baldpate, Inc.	viii
Boston Medical Laboratory	xii
David Memorial Nursing Home	viii
Electroencephalographic Laboratory, Inc.	xii
Glenside, Inc.	viii
Hoffmann–La Roche Inc.	After xii
Leary Laboratory	xii
Lederle Laboratories	iv, v
Thos. Leeming & Co., Inc.	vi
Eli Lilly and Company	Front Cover
Maltbie Laboratories, Inc.	xiii
Perkins School	viii
Picker X-Ray Corporation	vii
Riker Laboratories, Inc.	Back Cover
Ring Sanatorium	viii
Sanborn Company	xi
Schenley Laboratories, Inc.	ii
Sharp & Dohme (Division of Merck & Co., Inc.)	xvii
Smith, Kline & French Laboratories	After vi
Southern Medical Supply Co.	viii
E. R. Squibb & Sons (Division of Mathieson Chemical Corporation)	ix
Storrow House (Massachusetts General Hospital)	xii
Warner–Chilcott Laboratories, Inc.	iii
Washingtonian Hospital	viii
Westwood Lodge, Inc.	viii
Wilbur Imports, Inc.	viii
Winthrop–Stearns, Inc.	x
Wiswall Sanatorium	viii
Woodside Cottages, Inc.	viii

NOTICES *(Concluded from page 364)*

*11:00 a.m.–12:30 p.m. Hand Clinic (Physical Medicine and Rehabilitation Service). Boston City Hospital.
*11:00 a.m. Thoracic Conference. Cardiac Surgery in the Hypothermic State. Mr. Lance Bromley. Main Amphitheater, Peter Bent Brigham Hospital.

FRIDAY, SEPTEMBER 3
*8:00–8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*9:30–11:30 a.m. Medical and Surgical Grand Rounds. Drs. George W. Thorn and Francis D. Moore. Main Amphitheater, Peter Bent Brigham Hospital.
*10:00 a.m. Tuberculosis Surgical Clinic. South End Health Unit, 57 East Concord Street.
10:30–11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*1:30 p.m. Fertility and Endocrine Clinic. Free Hospital for Women, Brookline.
*1:30 p.m. Tumor Clinic. Mount Auburn Hospital, Cambridge.
*4:00–10:00 p.m. Alcoholism Clinic. By appointment. Washingtonian Hospital, 41 Waltham Street.

SATURDAY, SEPTEMBER 4
*8:00–8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*8:30–10:00 a.m. Orthopedic Staff Conference, Boston City Hospital.
10:30–11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

MONDAY, SEPTEMBER 6
Holiday.

TUESDAY, SEPTEMBER 7
*8:00–8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*9:00 a.m. Geriatrics Clinic. Peter Bent Brigham Hospital.
10:30–11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*12:00 m. Pediatric Grand Rounds. Cheever Amphitheater, Dowling Building, Boston City Hospital.
*12:00 m.–1:00 p.m. Pediatric Rounds. Burnham Memorial Hospital for Children (Burnham 4), Massachusetts General Hospital.
*12:15–1:15 p.m. X-Ray Conference. Dr. Merrill Sosman. Main Amphitheater, Peter Bent Brigham Hospital.
12:30–1:30 p.m. Medical Journal Club. Joslin Clinic. New England Deaconess Hospital.
*5:00–6:00 p.m. Service Meeting Followed by Clinical Conference (Medical, Surgical, Obstetrical). Faulkner Hospital.

WEDNESDAY, SEPTEMBER 8
*8:00–8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
10:30–11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*11:00 a.m. Boston State Hospital Psychiatric Seminar. Reception Building Auditorium, 591 Morton Street, Dorchester.
*12:00 m.–1:00 p.m. Clinicopathological Conference. Jimmy Fund Building, Children's Medical Center, 35 Binney Street.
*12:00 m.–1:00 p.m. Roentgen Diagnostic Conference. 5th floor teaching unit, Beth Israel Hospital.
*12:30 p.m. Weekly Pathological Meeting. Joslin Auditorium, New England Deaconess Hospital.
*12:30–1:30 p.m. Weekly Staff Review of Autopsies. Dr. William A. Meissner. Joslin Auditorium, New England Deaconess Hospital.
*1:00 p.m. Alcoholism Clinic. Peter Bent Brigham Hospital.
*1:00 p.m. Massachusetts General Hospital Hand Clinic. Out-Patient Department.
*2:00 p.m. Rectal Clinic. Peter Bent Brigham Hospital.
*2:00–3:00 p.m. Pediatric Conference for Practitioners. Jimmy Fund Building, Children's Medical Center, 35 Binney Street.
*4:00–5:30 p.m. Fracture Lecture, Boston City Hospital.
*4:00–6:00 p.m. Overholt Thoracic Conference. Joslin Auditorium, New England Deaconess Hospital.
*6:00 p.m. Alcoholism Clinic. Peter Bent Brigham Hospital.

*Open to the medical profession.

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